AN INVESTIGATION INTO AN ACUTE OUTBREAK OF 'CENTRAL NEURITIS'

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WITH SIX PLATES

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I. INTRODUCTION

While the epidemic shortly to be described was in progress, and before the specimens which had been taken to elucidate the morbid anatomy of the condition had been examined, the disease was spoken of as the 'Spanish Town epidemic,' because the majority of cases occurred in the neighbourhood of Spanish Town (St. Jago de la Vega), the ancient capital of Jamaica.

Subsequent enquiries, however, have shown that the condition is also met with in other parts of the island, and, moreover, there is a considerable weight of evidence to support the contention that the 'Spanish Town epidemic' represents the acute stage or an acute onset of a disease which, when it has become chronic, has for a long time been designated in Jamaica as 'Peripheral Neuritis.'

II. GENERAL HISTORY

Nearly all the patients are adults. Of a large number of cases reported to and seen by me there were but three children, and they were far from typical in the symptoms they exhibited; in fact, it is open to doubt whether they were instances of this condition at all. The youngest patient coming under my personal observation, undoubtedly a case of the condition under review, was a girl of 14 years of age (V.McC., B. 2 (*b*) of the series described later, p. 120).

All of those attacked during the epidemic were of the peasant class, that is, natives who worked as labourers on the sugar estates or who in rare instances had small holdings of their own. Males and females were equally affected. The epidemic started during the cutting and carrying of the cane crop, and the reporting of fresh cases ceased almost abruptly as soon as the crop was finished.

The following is a brief general description of the main features of the condition; differences shown by individual cases will be noticed in the detailed account of such.

In practically every instance the first symptom complained of is a sensation of 'itching in the eyes.' This comes on with comparative suddenness while the patient is at his usual work. In some cases both eyes are attacked about the same time, in others one eye is affected alone at first, and after an interval of varied length, from a few (three to four) hours to as many days, similar sensations are felt in the other eye. At this early stage the conjunctiva is congested and there is photophobia, but not of much intensity. Within the next three days or so the conjunctiva, both ocular and palpebral, is in a swollen, red, oedematous condition, the edges of the lids show abrasions, and small superficial ulcers form with discharge of pus.

Within four days to a week of the onset of the eye symptoms, a burning sensation in the mouth is complained of. This is referred to the mucous membrane of the lips and cheeks, but not as a rule the tongue. The lining membrane becomes red and inflamed and aphthae make their appearance, especially along the edges of the mucous membrane of the lips. At the angles of the mouth a small ulcer or fissure is often present. Salivation is not a common feature.

I noticed it only once in the twenty-one cases detailed, and did not observe it in any of the other patients seen at the hospital, although I saw more than a hundred suffering from this disease.

The soreness of the mouth gives rise to pain on eating for the first twenty-four hours or so; after that, in spite of congestion and ulceration, food is taken without any difficulty or complaint. This soreness is by the patients themselves often attributed to eating sugar-cane.

The affection of the eyes was usually treated by yellow oxide of mercury ointment, and the conjunctivitis cleared up fairly readily. For the stomatitis a mouth-wash containing chlorate of potassium and boric acid soon gave relief.

The above was the sequence of events at the onset in almost every case. One patient stated that the mouth was affected before the eyes, but this was the only exception.

No further symptoms develop during the succeeding week or so, in other words till about fourteen days after the first onset with itching sensations in the eyes. After this interval, however, further symptoms declare themselves, and the cases may be readily placed in one of two categories.

1. *Those with Diarrhoea and Intestinal Symptoms*

These patients have loose actions, increasing in frequency to as many as twenty-five in the day. Of those so affected, some die in a few days apparently from exhaustion, others slowly recover. No treatment seems to benefit the diarrhoea, which appears rather to cease gradually and spontaneously in those who recover. In the latter no further symptoms occur and recovery seems to be complete. The stools are watery and brown in colour.

2. *Those with Nervous Symptoms*

These patients are invariably constipated. As far as I myself observed, and from the histories of a large number of cases reported to me by the medical officer in charge of the hospital (Dr. Redwood White), in all of those exhibiting nervous symptoms constipation was the rule, and, as a corollary to this, in none of the cases belonging to the preceding class (intestinal cases) did any nervous symptoms develop.

The following gives a general résumé of the progress of 'nervous' cases:—

The patient states that he feels a sensation of numbness and tingling, starting in the toes and soles of the feet, occasionally accompanied by a feeling of heat and burning. The numb sensation slowly extends over the dorsum and up the legs to the knees—in some patients to the hips. Both limbs are affected together, and the spread is equal in both; in other words the legs appear to be affected segmentally, symmetrically, and simultaneously.

Some patients state that they feel 'pain in the knees,' but this is only complained of when movement of the joint is carried out. Palpation is quite painless, and there is no heat, redness, or swelling, in fact no objective sign of any joint trouble. With the spread of the numbness walking begins to be impeded, and in the course of three or four days, when the condition has extended to the knees, walking is impossible. The patient can no longer stand unless supported; there is marked incoordination and the patient has practically no control over the lower limbs. When supported and assisted in getting out of bed, the legs are thrown about with wild, exaggerated movements. In some cases, in the intermediate stages between the 'delicate,' unsafe gait of early numbness and the later total inability to walk, the gait is suggestive of that of tabes. Also at this stage there is no real loss of power, the knee-jerks and other deep reflexes are quite abolished, Babinski's sign gives no response normally in a native owing to the horny thickness of the soles caused by walking barefoot. Sense of position is not always, or even often, defective, although spontaneous disposition of the limbs is no longer possible.

In spite of the general complaint of numbness over so large an area, no alteration of sensation could, as a rule, be detected objectively. With the eyes bandaged the responses to finger-touch, cotton-wool, pin-head, and pin-point were correctly estimated and localised. The differentiation between heat and cold, even with a fairly wide range of temperature, was frequently defective. Although no change of sensation, at least no recognizable blunting of the sense of touch, was observable objectively, I noticed more than once in those who were still able to walk that one or both slippers might come off the feet and yet the patient would continue

his progress down the ward without them, or with one on and one off, and not notice the loss until he happened to look down and discover that he had left one slipper behind, when he would return for it.

The difficulty of walking was not made worse by closing the eyes, nor, again, did the patients watch their feet to help their progress. Some cases remained in this condition, but in others a similar sensation of numbness was complained of, after a further interval of one to four days or more, in the finger-tips, spreading rapidly over the palms; while, in a few, the backs of the hands, the forearms, and occasionally the upper arms, were affected also.

In the worst cases there was some difficulty of speech, due, as the patients described it, to numbness of the tongue; in fact, the condition was one of dysarthria and not aphasia. No numbness of the face was mentioned by any of the patients. No alteration of sensation could be detected in upper limbs or tongue by objective tests.

In those cases which terminated fatally, after a considerable period—four to six weeks or more—there was a marked general emaciation; no localised wasting of muscles could be detected at any time. The reaction of degeneration was not found in any instance, though in some there appeared to be a slight alteration in the nature of a less brisk response than normal, but nothing very tangible could be made out.

Some of the patients with nerve symptoms complained, during the early stages, of 'pain in the stomach,' and described this 'as if someone was pulling a rope tight round the chest.' It was clearly of the nature of girdle-pain, and only occurred in the second group of patients—those with nervous symptoms—not in any of those with diarrhoea and intestinal symptoms. This pain was not aggravated by food, in fact, the patients ate well without any discomfort.

The last stage of the fatal nervous cases was always the same. About forty-eight to seventy-two hours before death, when the patients were lying helpless in bed, diarrhoea would set in, and the exhaustion from the combined inanition, emaciation and diarrhoea soon brought about the fatal issue. Even towards the last, though the patient lay helpless and to all appearances totally paralysed, nevertheless there was not in reality a condition of true paralysis,

all movements could be performed, though feebly on account of the emaciation and general state of exhaustion.

Certain residual symptoms were noticeable in some of those who recovered after a considerable period of illness. These were in the main :—

1. Dimness of vision, usually spoken of as 'a darkness in front of the eyes.'

2. A certain degree of deafness, which usually increased as time went on, although the other symptoms might clear up almost completely.

3. A peculiar steppage gait, but without drop-foot, an exaggeration of movement of the legs but without the tabetic stamp; not, as a rule, with a wide base.

III. DESCRIPTION OF INDIVIDUAL CASES

A. INTESTINAL FORM

1. *Mild cases with recovery.*

(*a*) D.J., male, aged 35 years. This man was first taken ill on March 14th, 1917, with 'burning and itching of the mouth and tongue' and sores at the angles of the mouth. For the first two days the mastication of food was painful, so that he could only take soft, semi-fluid food in the form of 'pap' (cornmeal, arrowroot); after that time he could take ordinary food without any pain or discomfort. A week later, March 21st, his right eye began to "burn and itch" and four days afterwards the left eye was similarly affected. He stated he had been at work earning fair wages, and had been having 'plenty of good food.' He did not complain of any numbness or tingling and on examination showed no evidence of any nervous symptoms. There were no physical signs of any disease in heart, lungs, or abdominal organs. The bowels were inclined to be loose, but not very frequent, three or four motions daily. The urine was normal, the faeces showed the presence of ova of ankylostoma, ascaris, and trichiuris. When again examined on April 12th his condition was practically the same, and no nervous symptoms had developed. By the 25th he was much improved, mouth and eyes were well, no symptoms referable to the nervous system had appeared, and the patient left the hospital quite well a day or two later.

This was an exceptionally mild case and the mouth was attacked before the eyes, thus differing in onset from the rest of the cases, but otherwise the initial symptoms were typical. The presence of helminthiasis is of little significance, for the vast majority of the labourers in Jamaica harbour them, as reports made by me officially on various occasions during the last five years have shown.

Examination of blood from this case, carried out on March 28th, yielded the following results :—

Erythrocytes 3,940,000 per c.mm. Occasional nucleated red cells seen; no abnormalities in shape, slight degree of anisocytosis. Haemoglobin 70 per cent. Colour Index 0·89.

Leucocytes 8,400 per c.mm. Differential count of these gave:—

Polymorphonuclears			54·4 per cent.
Myelocytes			1·0 ,,
Metamyelocytes			0·2 ,,
Eosinophiles			3·8 ,,
Basophiles			0·4 ,,
Large Mononuclears			1·0 ,,
Transitionals			0·6 ,,
Lymphocytes: large		4·6	
small		32·8	
Rieder type		1·2	
		——	38·6 ,,

An Arneth count on this blood gave:—

I	II	III	IV	V	S (Stabkernige)
9·9	40·4	32·4	12·2	2·2	2·9

Arneth Index 66·5.

On April 12th 1917 there was a reduction in the percentage of polynuclear leucocytes with an increase in the eosinophiles and the large mononuclears; the details of the count on that date were:

Polymorphonuclears			43·0 per cent.
Promyelocytes			0·2 ,,
Myelocytes			1·2 ,,
Metamyelocytes			—
Eosinophile Myelocytes			0·4 ,,
Eosinophiles			9·2 ,,
Basophiles			0·8 ,,
Large Mononuclears			3 6 ,,
Transitionals			1·6 ,,
Lymphocytes: large		7·6	
small		29·8	
Türk's		1·0	
Rieder type		1·6	
		——	40·0 ,,

An Arneth count on this date gave:—

I	II	III	IV	V	S
3·7	36·4	35·3	13·9	3·2	7·5

The Index being 57·7.

Lastly, on April 25th the polymorphonuclears showed a still further reduction relatively, and the large mononuclears an increase, the remainder being almost the same as when the count was made on April 12th :—

Polymorphonuclears		36·6 per cent.	
Promyelocytes		0·4	,,
Myelocytes		0·8	,,
Metamyelocytes		0·6	,,
Eosinophiles Myelocytes		0·4	,,
Eosinophiles		9·8	,,
Basophiles		2·4	,,
Large Mononuclears		4·8	,,
Transitionals		3·8	,,
Lymphocytes : large	7·4		
small	30·4		
Türk's	0·2		
Rieder type	2·4		
	——	40·4	,,

Arneth count :—

I	II	III	IV	V	S
6·5	37·1	32·2	13·8	3·6	6·8

Arneth Index 59·7.

(*b*) J.S., male, aged 44 years. This man stated he was quite well and at work until March. 22nd, 1917, when his right eye began to 'itch and burn.' The following day the left eye was similarly affected and his mouth was sore, but the latter was at no time severe enough to prevent him from taking food.

When seen by me on March 28th the right eye was a little congested, like a case of ordinary catarrhal conjunctivitis ; the left was in a more severe state of inflammation, especially towards the inner canthus which was swollen and deeply injected.

He was inclined to have loose actions of the bowels for the previous two or three days. No nerve symptoms of any kind complained of or discovered on physical examination. The condition of the eyes and mouth remained the same for ten days or so, and then improvement set in, and when examined again on April 12th the right eye was normal, the left still a little injected. The diarrhœa had increased somewhat and he was having five or six loose actions daily. Still no nerve symptoms present. By the 25th the eyes and mouth were quite well, the diarrhoea had ceased, usually only one motion a day, occasionally two, but not loose. Examination of the urine of this patient showed that with the exception of some amorphous urates this excretion was normal. The faeces when first seen contained some pus and red corpuscles. A few cysts of *Entamoeba tetragena* were present, and cultivation yielded a growth of *Bacillus pyocyaneus*. Also while making a differential count of the leucocytes in this patient's blood a malarial crescent was seen ; no intracorpuscular parasites detected. In spite of all these things the temperature never rose above 100° F. He left the hospital quite recovered on April 28th.

A differential count of this patient's leucocytes yielded the following results on March 28th, 1917.

Polymorphonuclears			46·4 per cent.
Promyelocytes			1·2 ,,
Myelocytes			2·2 ,,
Metamyelocytes			1·0 ,,
Eosinophiles			2·2 ,,
Basophiles			1·4 ,,
Large Mononuclears			4·0 ,,
Transitionals			2·4 ,,
Lymphocytes: large	5·4		
small	30·8		
Türk's	0·8		
Rieder type	2·2		
	——	39·2	,,

Arneth count :—

I	II	III	IV	V	S
15·1	44·4	25·8	8·2	2·2	4·3

Arneth Index 72·4.

A second examination on April 12th revealed very little change :—

Polymorphonuclears			47·2 per cent
Promyelocytes			0·6 ,,
Myelocytes			2·8 ,,
Metamyelocytes			1·6 ,,
Eosinophiles			0·4 ,,
Basophiles			1·2 ,,
Large Mononuclears			3·6 ,,
Transitionals			0·8 ,,
Lymphocytes: large	6·0		
small	31·4		
Türk's	1·0		
Rieder type	3·4		
	——	41·8	,,

Arneth count :—

I	II	III	IV	V	S
13·6	50·4	22·0	7·6	1·3	5·1

Arneth Index 75.

2. *Severe case of intestinal form.*

J.A., male, aged 30 years. In normal health until April 2nd when his eyes started to itch, and within 24 hours became red and inflamed and he could not bear the light. During the succeeding 48 hours the lids became sore and 'matter stuck them together during sleep,' but in the day-time there was very little discharge. A few days later—four or five, so far as he remembered—the mouth became sore. The bowels had been acting normally all this time and until the 18th when the actions became loose and frequent. On account of the soreness of eyes and mouth and the diarrhoea, he presented himself at the hospital on April 22nd and was admitted.

When seen on the 25th the eyes were much better; there were no abrasions of the lids, but still some muco-purulent discharge in the folds of the conjunctiva and at the inner canthus. The general stomatitis had subsided, but there were small ulcers at the angles of the mouth. Bowels were still loose: he had had 7 to 11 actions daily since admission.

There were no nerve symptoms complained of, and none detected on minute examination. There was no history of any Yaws. The pus from the eyes yielded colonies of staphylococcus, micrococcus catarrhalis, and some Gram-positive rods of diphtheroid arrangement. The cells showed no inclusions of any sort. The blood was taken and a Wassermann reaction carried out, but the result was totally negative. Lumbar puncture was performed and cultures of the spinal fluid put up on various media—agar, nutrose-ascitic-agar, hydrocele-agar, blood-agar, and blood-serum (Löffler)—but all remained sterile and nothing abnormal was discovered in smears made from the fluid after centrifugalization; there was no deposit. Albumen not in excess.

The blood-count in this case gave the following results :—

Polymorphonuclears			56·6 per cent.
Promyelocytes			1·0 ,,
Myelocytes			1·2 ,,
Metamyelocytes			1·2 ,,
Eosinophiles			3·2 ,,
Basophiles			1·8 ,,
Large Mononuclears			3·0 ,,
Transitionals			2·2 ,,
Lymphocytes : large		6·2	
small		21·2	
Türk's		0·6	
Rieder type		1·8	
		——	29·8 ,,

Arneth count :—

I	II	III	IV	V	S
17·0	40·9	25·5	7·4	5·0	4·2

It will thus be seen that when the patient was at his worst the Arneth Index was unduly high, viz., 70·6. I regret that this patient left before another opportunity arose for taking his blood again.

Before passing on to a detailed description of the nervous form of this condition, the following case deserves mention because in the early stage it was like the nervous variety and during that time was accompanied by constipation, but later developed intestinal symptoms with total arrest of the nervous system affection and terminated fatally in a manner similar to that already related. The case was reported to me by Dr. White; it did not come under my personal observation. The history is not so complete as one would wish, as no details of the nervous system condition were stated. It is given here for what it may be worth, as linking the first group with the reports of the nervous cases which follow.

C.D., female, aged 23 years. In the early days of April 1917 her 'mouth became sore and ulcerated.' On the 11th she felt numbness and tingling in the toes and feet, gradually extending up the legs. On April 25th, when she could no longer walk unaided, she was brought up to hospital and was admitted. During the first few days she had loose but not frequent actions of the bowels, while the fortnight prior to admission (11th—25th, the period of spread of the tingling and numbness) she had been very constipated. Temperature on admission was 101° F.; it never rose above this and on the 25th had fallen to 99° F.; subsequently until her death on May 4th the temperature remained normal.

She lived for nine days after coming to hospital, and during this time there was no extension of the numbness or tingling and no further development of any

fresh nerve symptoms; diarrhoea, however, replaced the constipation, the number of actions varying from five to eleven daily, loose, brown and watery. No treatment seemed to be of any avail for this, and the patient died, as stated above, on May 4th.

B. NERVOUS SYSTEM CASES

1. *Mild with recovery.*

H.B., female, aged about 30 years. Towards the end of February, 1917, according to the history given by this patient, her left eye started to itch and became inflamed so that she could not bear the light. A week later the right eye was attacked with 'watering and burning.' Some four days later her 'mouth became sore.' On March 12th she first felt 'cramps' in her feet. Questions elicited the fact, in this and several other patients subsequently, that 'cramps' meant tingling and numbness, and nothing resembling what one usually understands by the term cramps. The toes were first affected and during the succeeding week these sensations extended as far as the knees. Both lower limbs were affected together. She had suffered greatly from constipation since the first week in March; up to that time she had always been very regular.

This patient was first seen and examined by me on March 28th, the third day after her admission to hospital. Her eyes and mouth were inflamed, but no ulcers could be discovered. There was distinct photophobia. She walked 'delicately,' not with a gait characteristic of any known condition, but most resembling one with corns or sore feet. She stated that the ground 'felt soft.' The knee-jerks were present, no loss of power could be detected in any of the leg movements. In spite of the complaint of numbness, by objective tests sensation appeared to be normal—wisp of cotton-wool, pin-head, pin-point, light finger-touch all felt and accurately localised with eyes bandaged. There was, however, some defective appreciation of difference between heat and cold. About six times out of ten heat was described as cold, never *vice versa*. All the reflexes were normal; no Romberg's sign, no ataxia, no loss of sense of position. The urine was normal; the faeces contained ova of ankylostome and ascaris. All her symptoms improved rapidly and she was able to leave the hospital on April 7th.

2. *Slightly more severe cases with recovery.*

(*a*) E.S., female, 27 years of age. Working and in normal health until March 14th when she noticed suddenly a 'burning and itching' in her eyes; both eyes were attacked almost simultaneously. There had been no soreness of the mouth. No further symptoms developed during the next ten days, but on March 24th she began to have a sensation of numbness in her toes; this numbness had extended to the knees by the 28th and she stated that the knees were painful, but only on movement of full extension or flexion. She complained also of 'pain in the chest as if a cord were tight round the stomach.' (The 'chest' of the native is below the belt). Her bowels had been obstinately constipated throughout the illness.

State when examined on March 28th: In spite of the extensive area of numbness complained of, no alteration of sensation could be detected by objective tests. Deep reflexes were absent, the superficial were variable. On standing there was distinct ataxia, more of a swaying character and a tendency for the legs

to double up, although there was no apparent loss of power in any of the leg movements when she was lying down or sitting on the edge of the bed. The patient was just able to walk alone, but for safety slight support was given; the gait was not tabetic (the medical officer stated that it had been when the patient first came in), but there was an irregular swinging of the legs and feet. The patient stated on this occasion that the knees were painful when at rest as well as on movement, but examination revealed nothing wrong, no swelling, no heat, no pain on palpation, and the movements of the joint were free.

The urine contained some amorphous urates and crystals of calcium oxalate, but was otherwise normal. The faeces contained ova of ankylostoma and trichiuris.

Blood-smears were taken and the following differential count obtained :—

Polymorphonuclears			27·2 per cent.
Promyelocytes			0·8 ,,
Myelocytes			0·8 ,,
Metamyelocytes			0·6 ,,
Eosinophiles			5·0 ,,
Basophiles			0·6 ,,
Large Mononuclears			3·2 ,,
Transitionals			1·6 ,,
Lymphocytes :	large	7·4	
	small	49·8	
	Türk's	—	
	Rieder type	3·0	
			60·2 ,,

The Arneth count on the same specimen gave :—

I	II	III	IV	V	S
6·6	38·9	29·5	16·9	4·4	3·7

Arneth Index 60·2.

When seen again on April 12th her condition was certainly improving; she could walk alone with much less difficulty. She stated that the tightness in the chest had almost gone, and her eyes were practically well. She left hospital ' feeling quite well again ' on April 16th and the medical officer states that he could not make out any residual effects of the illness. The very low percentage of polymorphonuclear leucocytes during the height of the illness is worthy of note.

(*b*) V.McC., female, aged 14 years, daughter of the case next to be related. This girl lived with her mother, but was not taken ill until about a month after the onset of the mother's attack. She stated that she could not remember whether there had been anything wrong with her eyes, but she knew that towards the end of March her mouth became sore and ulcerated, and during the next five to seven days eating was painful and difficult. A week after the mouth became sore she noticed a numbness of the feet, and during the succeeding week this sensation spread up as far as the knees; it never extended beyond this, and her hands were not affected at all. She was somewhat constipated but not markedly so. After these symptoms had persisted in practically the same condition, neither better nor worse, for a month, a gradual improvement set in and now (June 22nd) she ' feels much better.' Bowels regular.

On examination on this date she was able to walk without any difficulty, and she stated that ' the numbness had nearly gone but not quite.' Nothing abnormal detected on testing with wool, pins, touch, etc., as in the other cases. The only symptom which was giving any trouble was a ' darkness of the eyes.' This appeared to mean a haziness of vision and was mainly present when the patient

went out of doors into the bright sunlight. She noticed it very little while in a room, away from any glare, but stated that when she tried to read, the print after a little while tended to become blurred and the letters to run together.

Reaction to convergence seemed to be a little sluggish, but nothing else abnormal could be detected. Ankylostoma ova present in the faeces.

Smears of her blood were taken for subsequent examination and gave the following results :—

Four erythroblasts were seen while counting the 500 leucocytes, and three benign tertian malarial parasites.

Differential leucocyte count :—

Polymorphonuclears	... ...	37·4	per cent.
Promyelocytes	... ...	0·8	,,
Myelocytes	... ...	1·6	,,
Metamyelocytes	... ...	1·2	,,
Eosinophiles	... ...	8·2	,,
Basophiles ...	... ...	0·4	,,
Large Mononuclears	... ...	4·6	,,
Transitionals	... ...	2·8	,,
Lymphocytes : large	... ...	7·2	
small	... ...	33·8	
Türk's	... ...	1·8	
Rieder type	... ...	0·2	
		——	
Arneth count :—		43·0	,,

I	II	III	IV	V	S
6·4	42·8	38·0	4·8	1·1	6·9

Arneth Index 68·2.

The large mononuclear increase may be ascribed, in part at least, to the malaria present. The relative eosinophilia was probably due to the presence of ankylostomes.

3. *Moderately severe case with partial recovery ;* that is, recovery of general health, but with residual symptoms.

A.B., female, aged 33 years, mother of the patient last described. The history in this case was not remembered very accurately in detail, but her illness had begun four months prior to my seeing her. She was taken ill one day towards the end of February with soreness of the eyes and mouth. The eyes were first attacked with burning and itching ; the mouth shortly afterwards. The patient could not remember whether the eyes were attacked together, but, if not, the interval was short ; nor could she state what length of time elapsed before the mouth became involved. About a week after the latter, however, she felt a sensation of numbness in the feet, spreading gradually up the legs to the hips. In a few days more the hands became numb, the fingers being first affected, then the palms before the backs.

This condition remained practically unchanged for two months or so. At the beginning of May a gradual improvement set in, the hands regaining their normal feeling first. Ability to walk began to return towards the end of May, and when seen on June 22nd she was able to walk fairly well and stated that the numbness had nearly gone and she ' felt natural ' except that her eyes were ' a little dark.' Questioning her more exactly as to what she implied by this, I was told that objects in the bright sunlight were dim, and when she was indoors she found sewing difficult and reading still more so.

The bowels had been obstinately constipated all through the illness. No alteration of sensation could be detected by the usual tests; the knee-jerks were absent, and the walk was suggestive of 'steppage gait' but without drop-foot or with very slight drop. The stools contained hookworm ova in considerable numbers. Examination of the blood of this patient showed the presence of the ring forms of *Plasmodium falciparum*; occasional erythroblasts were seen, three during the counting of 500 leucocytes. The differential count of the latter gave :—

Polymorphonuclears		33·8	per cent.
Promyelocytes		0·8	,,
Myelocytes		1·2	,,
Metamyelocytes		1·0	,,
Eosinophiles		18·0	,,
Basophiles		0·6	,,
Large Mononuclears		6·6	,,
Transitionals		4·6	,,
Lymphocytes : large	9·2		
small	21·8		
Türk's	1·8		
Rieder type	0·6		
	——	33·4	,,

Arneth count :—

I	II	III	IV	V	S
3·0	39·1	37·3	11·8	3·5	5·3

Arneth Index 60·7.

The increase in eosinophiles is probably ascribable to the fact of the patient harbouring ankylostomes, the increase in mononuclears and transitionals to the malarial condition (some of the monocytes showed pigmentation). Apart from these the low polymorphonuclear percentage is worthy of note and may be compared with that of the preceding case (her daughter).

4. *Severe case with recovery of general health but with residual nerve symptoms.*

(*a*) E.M., female, aged 36 years. Her illness began suddenly 'about the middle of March,' with pain and itching in the eyes; the pain was of a burning, smarting character, and she was unable to bear the daylight. Both eyes were affected together, and in a few hours they became bloodshot. Four days later her mouth became sore, and for 24—48 hours eating caused pain, but after that she was able to take her ordinary food (cane, at that time, and some yam) with little if any discomfort.

On March 29th her feet began to feel 'burning and numb'; both were affected together. This sensation started in the toes and soles and gradually spread upwards to the knees or a little above. On April 5th the same numb sensation began in the finger-tips and when seen a week later, on April 12th, the palms were affected also, and she thinks 'the backs of the hands do not feel quite right.' She also complained on this day that her 'chest felt tied up.' On examination :—Her walk was not characteristic of any definite condition, the gait may rather be described as stiff and stilted and was not tabetic. She stated that the ground felt 'like india-rubber.' The knee-jerks were absent, superficial reflexes mostly normal, but varied a little at different testings. Babinski's sign not obtained (skin of sole very thick), but on deep pressure the whole limb was drawn up, no dorsiflexion of the toes noticed. No alteration of sensation could

be made out by the usual tests. Examination of the cerebro-spinal fluid revealed nothing abnormal, very few cells were present, the majority were mononuclears; no growth was obtained from attempts at culture on various media; albumen not in excess. Wassermann reaction carried out with the fluid was negative, as also was the test with her blood-serum.

The other results of the blood examination were :—

Polymorphonuclears			37·4 per cent.
Promyelocytes			0·4 ,,
Myelocytes			1·6 ,,
Metamyelocytes			0·6 ,,
Eosinophiles			4·2 ,,
Basophiles			0·6 ,,
Large Mononuclears			4·6 ,,
Transitionals			1·8 ,,
Lymphocytes : large	9·8		
small	35·6		
Türk's	1·0		
Rieder type	2·4		
	——	48·8	,,

Arneth count :—

I	II	III	IV	V	S
9·1	53·0	21·4	9·6	3·2	3·7

Arneth Index 72·8.

No nucleated red cells seen; no malarial parasites, no pigmented mononuclears. No ankylostomes or ova of any helminth seen in the faeces.

This patient was seen again on April 25th; she stated then that the feeling of tightness of the chest was worse, but that the other symptoms had improved. She described the sensation in the feet then as being 'tightened up but not so numbed.' She could not explain more definitely what she meant by this, and I am not able to conjecture. She denied that the feeling at all resembled ordinary cramps. She walked quite as well, and, in my opinion, better and less stiffly than on the 12th.

The blood examination on this occasion gave results very little different from those of the 12th.

Polymorphonuclears			41·2 per cent.
Promyelocytes			0·4 ,,
Myelocytes			0·8 ,,
Metamyelocytes			0·6 ,,
Eosinophiles			4·0 ,,
Basophiles			1·2 ,,
Large Mononuclears			3·8 ,,
Transitionals			2·8 ,,
Lymphocytes : large	8·4		
small	33·2		
Türk's	1·6		
Rieder type	2·0		
	——	45·2	,,

Arneth count :—

I	II	III	IV	V	S
5·8	47·1	35·4	7·8	1·5	2·4

Arneth Index 70·6.

She remained in hospital for some time afterwards, but very little further improvement took place and she left. I was unable to find out her residence and therefore cannot state what progress, if any, took place subsequently.

(*b*) C.S., female, aged 30 years. This patient was admitted to hospital on April 14th, and gave the following history; early in April (she is not certain of the date) her eyes and mouth became 'itching and sore.' The eyes were both affected together a few hours before the mouth became involved. She suffered from constipation. No other symptoms made their appearance till April 12th when she felt her toes and feet becoming numbed; as this sensation continued to spread she came to the hospital, as already stated, on the 14th. Since that date the condition has extended to the knees, and when seen on the 25th she said that in addition to these symptoms in the legs she had a 'sense of tightness round the chest.' 'On examination it is noticed that she can walk fairly well, lifting the feet as in a modified steppage gait, but requires some support, saying that she feels unsteady owing to the ground feeling soft. Knee-jerks are absent; Romberg's sign is not present; though unsteady in walking, she can stand fairly well, and closure of the eyes does not affect this. Pupils react normally to light and convergence. By objective tests no alteration of sensation is made out in the numb area, and there is no hyperaesthetic zone at the level of the girdle sensation.'

Cerebro-spinal fluid was taken, smears made and various culture-media inoculated with the fluid, but no growth occurred, and nothing abnormal was seen in the original smears, nor in those made after centrifugalization of the fluid.

The Wassermann reaction was carried out with both this fluid and the blood, but the result was totally negative with both.

Blood examinations gave:—

Polymorphonuclears		43·0	per cent.
Promyelocytes		0·2	,,
Myelocytes		0·8	,,
Metamyelocytes		0·8	,,
Eosinophiles		4·0	,,
Basophiles		1·8	,,
Large Mononuclears		3·6	,,
Transitionals		2·4	,,
Lymphocytes: large	11·6		
small	29·6		
Türk's	1·4		
Rieder type	0·8		
	——	43·4	,,

Arneth count gave:—

I	II	III	IV	V	S
6·5	42·3	25·6	13·0	3·7	8·9

Arneth Index 61·6.

No nucleated red cells seen, no malarial parasites or pigmented mononuclears. Faeces and urine normal, no helminth ova present in the former.

(*c*) R.H., female, aged 42 years. This patient gave the following history: She was quite well until May 23rd, 1917, when, while she was at work on the sugar estate, her right eye began to itch. The next morning the lids were stuck together on waking and the light caused pain. That day the left eye became similarly affected, and two days afterwards her mouth became sore and she could take food

only with pain and difficulty. White patches and superficial ulcers formed on the lips and tongue. On June 2nd, ten days after the onset, her 'feet began to burn'; the toes and soles of the feet were first attacked, but the sensation rapidly spread, reaching as high as the hips in another week; both were affected together and equally. Within 24 hours of onset the sensation of burning was replaced by numbness. On June 9th there was a 'tight feeling' round the lower part of the abdomen, and, by this date, walking, which had become increasingly difficult, became impossible and when attempting to walk she fell. She had been constipated from the beginning of her illness, though up to that time her bowels had always acted regularly. On June 10th her finger-tips felt numbed (without any preceding burning) and this sensation spread over the palms and dorsa as far as the wrists, but no further. Moreover, the affection of the hands was much slighter than in the feet and legs, and after persisting for a week, improvement in sensation in the hands set in.

I saw her on June 22nd, and found the hands were, as she stated, nearly well. She could pick up a pencil between a finger and thumb, except with the little finger of the right hand. The lower limbs had also considerably improved, and she was able to walk unaided, but slowly with a spastic gait, the feet, as it were, sticking to the floor. She stated that the ground felt soft. The knee jerks were not elicited, but the quadriceps was felt to contract when the patellar tendon was tapped. She was able to stand without difficulty with her eyes closed, and the walking was not made appreciably worse when they were bandaged. There were no signs at all of any pellagrous condition.

By the usual tests no alteration of sensation could be detected, except an irregularity in the interpretation of heat and cold. She stated that her eyes were 'a little dark'; this, she explained by saying that sewing was difficult (not on account of any numbness of the fingers, but owing to her not seeing the stitches clearly), and on attempting to read the letters ran together and the print became blurred. Though she did not complain of deafness, I am of opinion that there was a slight degree of difficulty of hearing, evidenced by her turning her head to concentrate her attention to the direction of the voice, when questioned. Her friends also thought she was 'a little hard of hearing since her illness.'

Her blood was taken and gave the following results of a differential leucocyte count :—

Polymorphonuclears			30·6 per cent.
Promyelocytes			0·4 ,,
Myelocytes			0·8 ,,
Metamyelocytes			0·2 ,,
Eosinophiles			15·4 ,,
Basophiles			0·4 ,,
Large Mononuclears			2·2 ,,
Transitionals			1·0 ,,
Lymphocytes: large	8·6		
small	39·0		
Türk's	1·0		
Rieder type	0·4		
		49·0	,,

Arneth count :—

I	II	III	IV	V	S
6·6	45·7	27·5	12·4	1·9	5·9

Arneth Index 66·05.

No malarial parasites were seen, nor any pigmented mononuclears; one erythroblast was observed in making the count. The eosinophilia is explicable by the fact that the faeces contained ova both of *Ankylostoma duodenale* and of *Ascaris lumbricoides*.

5. *Severe and chronic cases.*

(*a*) E.G., female, aged about 20 years. States that she suffered from an attack of Yaws as a child, but there are no signs of that disease to be seen now, unless an unpigmented patch and a scar on the right wrist are taken as evidence of this. The patient states that she was well until April 19th, when her left eye began to itch and soon after became red, and she could not bear the light with that eye. Some 24 hours later the right eye was similarly affected. The mouth was also sore, but the patient does not remember with certainty the exact chronological sequence of events at the onset. At one time she stated that there was an interval of a week between the eye and mouth involvements, at another that they were affected almost at the same time. She had not felt any pain on eating at any time since her illness began, not even when the mouth became sore.

A fortnight 'or thereabouts' after the onset—that is, on or about May 2nd—her toes and the soles of her feet began to feel numb, and, as she described it, 'like needles pricking and worms crawling there.' These sensations spread over the dorsum of each foot and at this time when she walked she 'felt as if walking up in the air.' Walking became increasingly difficult and on May 10th she fell down on the road. She had no convulsions or any loss of consciousness, as far as can be ascertained, and she got up immediately unaided and walked home. She took some time to get back, saying that her legs and feet 'felt heavy, but not tired.' After that date she no longer attempted to walk out of doors, and at home she experienced greater and greater difficulty in moving about. This inability progressed so rapidly that by the 13th or 14th May she had to move about her room on hands and knees, and finding that she could not climb into her bed, she had the bed made up on the floor. By this time the numbness, tingling and formication had extended to the hips. The trunk does not appear to have been affected, at all events she denied feeling any of these sensations there, nor was there anything suggestive of a girdle pain. On the 18th May she felt the tingling and numbness in the tips of the fingers of both hands, and by the 20th the palms were affected also; the backs of the hands were normal then, but have become involved since. There was never any sphincter affection at any time. Except for the 'creeping and pricking sensations' there had never been any pain. She had not felt any numbness of the tongue, nor had her speech been affected. She states definitely that she had no headache or backache, no pain or discomfort along the spine, no abdominal or thoracic pain, no 'rope-' or girdle-sensations, or giddiness.

She was first seen by me on May 23rd, when the above history was obtained, Now she says she feels no longer any pricking in her legs, feet, or hands; in fact as regards the hands she only has the feeling of numbness as far as the wrists, but in the feet and legs, in addition to this, she still has the 'creeping and crawling' sensations. For the first three weeks or so of her illness she suffered a good deal from constipation; but lately she has been having two or three loose actions daily and feels better, the numbness and other unpleasant sensations being a little less; she affirms this quite definitely.

The following were the conditions noted when the patient was examined on May 23rd:—No pigmentation of the hands, forearms, face or neck; nothing

suggestive of pellagra. There is a patch over the lower two inches of the radial aspect of the right forearm where the pigment is deficient, and in the centre of this is an old cicatrix. There is no roughening of the skin over the area. This may be the result of an old attack of yaws; she cannot state when she first noticed it and thinks it has always been there. Her mental state was good and she replied readily to all questions.

Muscular power. All the ordinary movements are performed with little, if any, diminution of power; there is certainly not more than would be accounted for by the ataxic condition present.

Sensation. The changes are mainly subjective, but there is a definite, though slight, alteration observed with the following tests. A wisp of cotton-wool is felt (with the eyes bandaged) all over the area where the patient states she has the numb sensation, but affirms that the feeling of the touch of the wool is 'not the same' as over the normal skin. Pin-head and pin-point are readily perceived and differentiated, and accurately located.

Heat and cold. These are distinguished correctly, but there appears to be a little delay in the perception. Thus, touch is replied to immediately, but heat and cold seem to be noticed only after an appreciable interval of two to four seconds.

Vibration sense. Is definite and appears to be quite normal.

Position and Joint sense. Do not seem to be affected. In one instance, with the right leg bent up and crossed over the left, she stated that the limb was straight, but since on every other occasion the position of the limbs was accurately described I am inclined to think that either she was describing the position of the left leg, or she was not attending, or lastly that she did not grasp the question, for this was in one of the earlier tests of position sense.

There is no pain on ordinary pressure of the muscles, no joint pain, either spontaneous or on movement or on palpation. There is no tremor on extension of the arms and fingers, nor when at rest. The pupils react normally, there is no nystagmus.

There is no hyperaesthesia above the anaesthetic or paraesthetic areas. The cranial nerves are not affected; sight, hearing, taste, all seem to be normal. She can appreciate the difference in thickness between two similar objects, e.g., two note-books, and also weights within 10 grammes of each other. She recognises a pen, a hairpin, a coin, a watch, by sense of touch with eyes shut.

Ataxia. When asked to touch the tip of her nose with the tip of her forefinger, or to bring the finger-tips together, all with the eyes closed, she usually missed the contact by a couple of inches or so; there is no intention-tremor. She can follow an object moved in space fairly closely with either hand. She has some difficulty in picking up a pencil between the thumb and first finger of either hand; this difficulty increases with the middle and ring fingers, and she is quite unable to pick it up between the thumb and little finger. With a smaller object, such as a pin, all efforts are ineffectual, she cannot pick it up even with the thumb and forefinger.

As regards the legs: she is quite unable to walk or even to stand unaided. Romberg's test, therefore, cannot be carried out. When she is supported on both sides and an endeavour is made to walk, the legs are thrown wildly about, or they cross over each other, or may double up under her.

When she lies in bed, however, she can, though badly, more or less follow a moving finger with her feet.

Reflexes. Deep reflexes are absent. No knee-jerk or Achilles' tendon-jerk elicited, nor the supinator or triceps reflex. The superficial reflexes are present; the plantar somewhat exaggerated. The Babinski reflex was certainly not extensor,

though, as already stated, in a native this is difficult to obtain at all, owing to the thickness of the skin of the sole. In this patient the foot and leg were drawn up sharply, but there was no dorsiflexion of the toes.

The temperature had generally varied between 98° and 99° F., and once touched 100° F., but since the 20th it had been normal. The blood and cerebro-spinal fluid gave a negative Wassermann reaction. The results of the detailed examination of the former will be given at the end of the description of her case. The latter was clear and to all tests normal; cells few, practically all mononuclears, no organisms, bacteria or treponemata found. Albumen not in excess.

The faeces contained ova of ankylostoma.

On June 11th she was examined again; her general condition seemed to be a little improved. She stated that the numbness in hands and wrists was less marked, and this must be taken according to her assertion, for, as shown above, tests with wool, etc., revealed nothing objectively before. The inco-ordination of the upper limbs remained as at previous examination; the condition of the legs was as before. They seemed to be no worse, but there was certainly no improvement. The position-sense was not quite so accurately estimated as on the former occasion; the muscle pressure sense was still unaffected. The stereognostic sense had distinctly deteriorated. With her eyes shut she could not recognize by touch, a key, a pocket-knife, a match-box, a coin, etc., although she readily named than on opening her eyes. Also, as regards weights (as stated above, closely approximating weights were distinguished at the examination on May 23rd), two match-boxes containing the one a 5 gramme weight the other 72 grammes were not distinguished with any accuracy. She usually said the box with 5 grammes was the heavier, or that they both felt the same. When the weights were hung on to the toes or round the ankles she made similar replies. She was still unable to walk or even to stand. The faeces still contained hookworm ova in considerable numbers.

A third examination carried out on June 22nd revealed nothing new. She still affirmed that the numbness of the hands and fingers was diminishing; and now added that, though the feet and legs felt as numbed as before, the tingling had disappeared, but the crawling sensations had not altered. On repeating the same tests as on the two previous occasions no change could be detected. She stated on this occasion that her eyes seemed 'a little dim' and that reading was difficult because the letters ran together.

I regret to say that this patient, showing no further improvement, was taken away from the hospital shortly afterwards.

Blood examination on May 23rd gave the following results:—

Erythrocytes 3,312,000 per c.mm. Occasional nucleated cells seen, some degree of anisocytosis, no noticeable poikilocytosis, staining reactions normal, no malarial parasites.
Haemoglobin 60 per cent. Colour Index 0·9.
White corpuscles 9,300 per c.mm.

Blood examination on June 11th gave:—

Erythrocytes 3,480,000 per c.mm.; none nucleated.
White corpuscles 9,000 per c.mm. Haemoglobin 60 per cent.
Colour Index 0·89.

On the 23rd June the erythrocytes numbered 3,600,000 per c.mm.; Haemoglobin 64 per cent., practically the same as on the previous occasion, giving a colour index of 0·88. Leucocytes 10,100 per c.mm. There is thus an increase

in leucocytes, and, as will be seen, a tendency to a thrust to the left in the Arneth count as the disease continued.

There follows in tabular form the results of the differential leucocyte counts on the three occasions :—

	May 23rd	June 11th	June 23rd	
Polymorphonuclears ...	37·8	46·4	49·0	per cent.
Promyelocytes	0·6	0·8	0·8	,,
Myelocytes	2·0	2·4	1·4	,,
Metamyelocytes	0·8	1·6	1·2	,,
Eosinophile Myelocytes	0·4	0·2	—	,,
Eosinophiles	10·0	8·4	5·8	,,
Basophiles	1·8	0·8	0·2	,,
Large Mononuclears ...	3·4	4·6	4·2	,,
Transitionals	3·4	3·4	2·2	,,
Lymphocytes	39·8	31·4	35·2	,,

Arneth counts :—

	I	II	III	IV	V	S
May 23rd	5·8	48·7	31·2	5·3	2·6	6·4
June 11th	8·2	46·1	28·9	7·3	3·5	6·0
June 23rd	11·4	58·0	19·2	2·0	1·6	7·8

Arneth Index :—

May 23rd 71·1 ; June 11th 68·7 ; June 23rd 79·0.

(*b*) C.H., female, aged 36 years. This patient stated that she was quite well until the end of April, 1917. She was doing some washing when quite suddenly she felt an itching in her eyes ; the right was first attacked and some three to four hours later the left. She said that her mouth was not sore at any time. The following day there was 'stickiness' of the lids, and the light caused pain. Three days later she felt that her toes were getting numbed and then the soles of her feet. During the succeeding week this feeling gradually extended up to the hips. On or about the 10th May similar numb sensations started in the fingers, but now accompanied by pricking ; this extended as far as the wrists but not beyond. She was very constipated throughout. The condition remained practically unchanged for a month, then from about 12 June she thought she had improved a little, but the improvement was very slight.

On examination on June 22nd I have the following notes :—

'Patient is unable to walk at all, or even stand without being supported. There is marked ataxia. There is also a considerable want of co-ordination in the movements of the fingers, but this had improved more than any other symptom apparently, and she is now trying to sew, but finds it very difficult and often drops her needle. She can pick up a pencil between the thumb and first, middle or ring finger, but not the little finger. With the left hand the attempt to do the last is better than the right (the patient is right-handed). She complains now of pains all down the arms, most troublesome at night. On further enquiry into this symptom I am given to understand that this sensation is not that of real pain, but that the arms feel uncomfortable and that she cannot place them in any position in which they are eased sufficiently for her to sleep well. Knee-jerks not elicited ; wrist-jerk doubtful. The changes, as in other cases, are practically entirely subjective, no true loss of sensation is detected over the " numb " areas by the usual tests. No ova detected in the faeces.'

A blood examination was made on June 22nd, and gave the following results :—

Polymorphonuclears	...	...	...	...	48·2	per cent.
Promyelocytes	...	...	...	...	0·6	,,
Myelocytes	...	...	...	...	1·4	,,
Metamyelocytes	...	...	...	...	1·4	,,
Eosinophiles	...	...	...	...	3·2	,,
Basophiles ...	...	...	...	...	1·6	,,
Large Mononuclears	...	...	...	...	2·4	,,
Transitionals	...	...	...	...	3·2	,,
Lymphocytes : large	...	...	...	9·6		
small	...	...	...	24·6		
Türk's	...	...	...	3·4		
Rieder type	...	...	...	0·4		
				——	38·0	,,

Arneth count :—

I	II	III	IV	V	S
8·7	46·5	30·3	7·0	4·6	2·9

Arneth Index 70·8.

It will thus be seen that the stage of the disease corresponds closely to that of the last case when the blood was taken on the third occasion, and the differential count is strikingly similar. The main difference is in the eosinophiles, and this is explicable by the presence of hookworms in the former and their absence from the latter.

6. *Severe case ; result not known, probably fatal.*

A.D., female, aged 25 years. The history obtained from this patient was that on March 1st, 1917, her eyes suddenly ' began to burn.' Previously she had been quite well. A week later her mouth became sore at the corners, but this did not cause any pain or interfere with her taking food. A fortnight after the onset (March 15th) she felt a sensation of ' heat in the soles and tops of the feet,' spreading up gradually till it reached the knees by the 21st. Coincidently with the extension of the feeling of heat the toes and soles of the feet began to be numb. Up to the 21st she was able to walk, but by the 23rd the numbness had extended to the knees and walking was impossible, owing, as she stated, to ' the knees not being able to bear her.' On the 24th her finger-tips began to feel numb (no sensation of heat here), and the following day the same sensation was spreading over the backs of the hands. On the 27th she stated that her ' face felt tight,' but not numbed at all. She had had plenty of food (mainly sugar-cane) prior to the onset of her illness ; she had been getting good wages and was not starved at all. There had been no vomiting, no fever, and she had been constipated throughout. Three days later she was much worse ; I was told that she had become ' totally paralysed,' but this turned out to mean that, owing to the numbness and inco-ordination when movement was attempted, she lay still ; no actual paralysis could be made out anywhere.

It is a matter of great regret that this case could not be watched through and an autopsy obtained, as the march of events in her case, though following the usual lines as regards the nature of the symptoms, was much more rapid than the average. As she was going downhill, her friends came and took her away at her own request, and subsequent enquiries at the address she gave proved fruitless, and she could not be traced.

Her state on examination on March 25th was as follows :—

She had to be supported on both sides when she was asked to get out of bed; there was marked ataxia, the legs being quite uncontrollable; the gait, therefore, could not be tested, nor Romberg's sign. There was no resemblance to the tabetic kick, the legs simply 'flopped about' uncontrolled. Pupils reacted to light and convergence. Knee-jerks not elicited, nor Achilles' tendon reflex, nor the wrist-jerk; the triceps jerk was obtained. When sensation was tested, cotton-wool, pin-head, pin-point, heat and cold were all correctly recognised and accurately localised everywhere. The only change detected was that perhaps once in seven times heat was described as cold, and that generally the conduction of this sensation appeared to be delayed. Allowance being made for the ataxia, no definite loss of power in the various movements could be detected. There was no involvement of the sphincters. No mental change; the patient was perfectly rational and replied to all questions clearly and promptly and stated that although even the wisp of cotton-wool was felt, it 'seemed duller' over the numb parts. Vibration-sense apparently normal, but sense of position and joint-sense were defective.

The urine contained some dicalcium phosphate crystals, but nothing else abnormal. Faeces showed ova of ankylostoma and ascaris, but unaccompanied by any eosinophilia.

Blood examination on March 25th, 1917:—

Polymorphonuclears			52·8 per cent.
Promyelocytes			1·4 ,,
Myelocytes			1·0 ,,
Metamyelocytes			1·0 ,,
Eosinophiles			2·0 ,,
Basophiles			0·6 ,,
Large Mononuclears			2·6 ,,
Transitionals			1·6 ,,
Lymphocytes:	large	7·6	
	small	28·2	
	Türk's	1·0	
	Rieder type	0·2	
		——	37·0 ,,

Arneth count:—

I	II	III	IV	V	S
10·3	42·0	29·9	11·0	3·4	3·4

Arneth Index 67·2.

While doing the count a few erythroblasts were seen, some with bizarre-shaped nuclei; some degree of chromatophilia, and two corpuscles showed basophilic stippling.

7. *Severe cases terminating fatally.*

(*a*) J.P., male, aged 25 years. History: The illness began on March 3rd with 'burning in the eyes' followed after an interval, the duration of which is not remembered, by 'sore mouth.' On March 17th his feet felt numbed and walking became difficult. During the succeeding three or four days the numbness extended up the legs and the 'knees felt hot,' but were not painful. The symptoms of numbness and heat have been slowly getting worse. When seen on March 28th the eyes were suffused, and the lids exuded a little pus; also there was some, not very marked, degree of photophobia. There were some

shallow ulcers, more of the nature of superficial abrasions, along the edges of the lips. Knee-jerks could not be elicited; gait showed little if anything abnormal, there was a tendency to lift the feet higher than normal, suggesting the steppage gait. Pupils reacted normally; Romberg's sign not present. No loss of power could be detected, nor, objectively, any alteration of sensation. No muscle-pain, the vibration-sense not affected, and weights readily differentiated.

Some pus was taken from the eye, and also smears from the surface and edges of the abrasions on the lips. Cultures on various media both aerobically and anaerobically revealed nothing abnormal; the various colonies which grew were examined, and only the usual air and mouth-organisms were found. No cell-inclusions could be discovered after prolonged search. The faeces contained ova of ankylostoma; urine normal.

On April 12th the condition was worse; he was by this time quite unable to walk, and his fingers, hands, and wrists felt numbed. His speech was inclined to be blurred, and he stated that his tongue 'felt heavy.' All tongue movements carried out normally, no sign of any atrophy of tongue muscles, or of any other muscles. Taste normal, but weights as wide apart as 10 and 70 grammes not differentiated with any accuracy. Joint and position-senses also affected. He did not move in bed owing to the numbness, but when asked to move he could do so normally though slowly, and there was no sign of actual paralysis anywhere. Quoting from my notes on this date:—He is becoming emaciated and does not take his food well. He states that he has no appetite, but he can swallow without any difficulty; there is no change in his voice. He has been very constipated from the beginning of his illness

He remained in the condition described above, except that he became generally weaker. On April 20th diarrhoea set in, and he had an average of eight loose watery actions daily. He lay quite helpless and passed all motions under him, but not strictly involuntarily; that is, he felt the desire, but the motion passed before the nurse could come and attend to him. The diarrhoea continued until the 25th when death occurred at 3.15 a.m. A post-mortem examination was performed the same day. Cultivation of the faeces during the last few days revealed nothing abnormal, with the exception of a chromogenic organism giving the following reactions:—

Markedly motile, rendering milk alkaline, but without the production of any clot; producing acid but no gas in the following media: lactose, saccharose, dulcite, mannite, glucose, maltose, and dextrin. Examination of the blood was carried out on three occasions, namely, on March 28th, April 12th and 18th.

March 28th:—

Erythrocytes 4,200,000 per c.mm.; two nucleated red cells seen, no malarial parasites detected.
White cells 8,600 per c.mm.; Haemoglobin 80 per cent.
Colour index 0·85.

April 12th:—

Erythrocytes 3,800,000 per c.mm.; no nucleated corpuscles seen.
White cells 9,600 per c.mm.
Haemoglobin 72 per cent. Colour index 0·95.

April 18th:—

Erythrocytes 3,600,000 per c.mm.
White cells 11,200 per c.mm.
Haemoglobin 62 per cent. Colour index 0·86.

Differential leucocyte counts on these three occasions :—

	March 23rd	April 12th	April 18th	
Polymorphonuclears ...	45·0	41·0	64·0	per cent.
Promyelocytes	0·4	0·6	1·0	,,
Myelocytes	0·6	1·4	1·2	,,
Metamyelocytes	1·0	0·2	—	,,
Eosinophile myelocytes ...	—	0·4	—	,,
Eosinophiles	4·0	7·2	6·8	,,
Basophiles	0·6	0·8	1·0	,,
Large Mononuclears ...	2·6	2·2	3·2	,,
Transitionals	2·2	1·8	2·6	,,
Lymphocytes—				
large ...	7·4	5·4	3·2	
small ...	35·2	35·8	14·6	
Türk's ...	1·0	2·8	2·2	
Rieder	—	0·4	0·2	
	43·6	44·4	20·2	,,

Arneth count :—

	I	II	III	IV	V	S
Mar. 23rd	4·5	33·8	40·0	12·5	5·4	3·8
Apr. 12th	6·8	36·6	36·1	12·7	3·4	4·4
Apr. 18th	6·6	43·1	30·6	9·7	6·9	3·1

Arneth Index :—

March 23rd 58·3 ; April 12th 61·4 ; April 18th 65·0.

It will thus be seen that as the patient got worse the polynucleosis increased and the lymphocytes decreased, and the Arneth count shifted more and more to the left ; although the final differential count shortly before death gave practically a normal percentage.

The cerebro-spinal fluid was taken by lumbar puncture, smears and cultures put up, but no growth occurred on any of the media employed, and nothing abnormal was detected in the smears.

At the post-mortem examination the following points were noted :—

The body was emaciated, the subcutaneous fat was only small in amount, but the muscles did not appear to be much wasted and were of a good colour. Spinal fluid was taken again, was clear and limpid, and flowed slowly, drop by drop from the lumbar puncture (cultural attempts proved sterile as on the former occasion).

Spinal cord. Vessels not injected, except a little perhaps over the lumbar region. There was no meningitis, nor did the cord itself appear softer than normal on section.

Brain. No inflammation of the meninges ; there was a faint serous haze over the vertex ; nothing else noticeable. There was very little fluid in the ventricles, and the brain tissue appeared normal on section.

Thorax. Practically no subcutaneous fat. There were firm pleural adhesions on both sides, more on the right and firmest there, especially over the upper lobes ; no fluid in the pleural cavities. No sign of any tuberculosis. The upper and middle lobes of the right lung were firmly bound together by adhesions, in fact they could not be separated without dissection. The lung tissue was crepitant all over.

Heart. Pericardium contained 10 to 15 c.c. of pale, straw-coloured fluid. There was a slight atheroma at the base of the aorta ; nothing else abnormal, valves competent.

Abdomen. Nothing abnormal seen, no free fluid, no signs of any peritonitis; stomach normal.

Spleen. A little enlarged, with slight local perisplenitis, tissue macroscopically normal.

Liver and *kidneys.* Normal.

Mesenteric glands. Enlarged and pink.

Small Intestine. Contained three ascarides. The contents otherwise consisted of a greenish material, no blood. The vessels were congested over the lower six feet or so, but not markedly till about two feet above the ileo-caecal valve, when the congestion became deep and there were small ulcerous patches with denudation of the mucous membrane (resembling a dysenteric condition) but the surface was pale, not injected. Here and there were small pigmented patches varying from one to four centimetres in diameter. Peyer's patches apparently normal.

Large Intestine. Congested and showed small denuded areas, mainly in the ascending colon and the hepatic flexure.

The following tissues were taken for further microscopic examination: Brain cortex, cerebellum, pons, medulla, basal nuclei, olfactory lobe, optic nerve, roots of the various cranial nerves; cervical cord at three different levels, dorsal and lumbar cord at three levels, lumbo-sacral cord; sciatic, median, and ulnar nerves; skeletal muscles, heart muscles, tongue muscle, lung, thyroid; liver, spleen, kidney, adrenals, stomach mucous membrane, intestine, and pancreas.

These were treated in various ways and will be spoken of presently, as the findings were practically identical in this and the case next to be described, and one description of the microscopical findings will suffice.

(*b*) G.P., male, aged 36 years. The history obtained from this patient was that he was quite well until Feb. 20th. During that day his right eye started to 'itch and burn,' and within a few hours the left was similarly affected and the light caused pain. He had been earning good wages and stated that he had had plenty to eat, though the food consisted largely of sugar-cane. On the third day of the illness (Feb. 22nd) his mouth became sore and for 24 hours or so he could not eat on account of the soreness; after the first day of this, however, he was able to take food with little or no discomfort. No further symptoms declared themselves until the beginning of March, the 2nd or 3rd, when he began to feel a numbness in the toes and soles of the feet; this gradually extended over the dorsa and up the legs, till by the 14th he had considerable difficulty in getting about. The difficulty increased, and by the 20th walking became impossible. Four days later he began to feel a similar numbness, with some tingling in the finger-tips of both hands. He had been obstinately constipated from the start.

When examined on March 28th he was lying in bed; he was not paralysed, but on attempting to get up, the ataxia was extreme, the legs being thrown wildly about; the arms were partially ataxic also. Deep reflexes (knee-jerk, ankle-jerk) not obtained in the legs, nor the wrist-jerk in the upper extremity; the triceps was doubtful. Testing the various movements of arms and legs revealed the fact that there was very little, if any, loss of power; but the joint and position senses were defective. As regards sensation nothing could be detected objectively except some confusion between heat and cold. Although no defect in perception of sensation to touch, pin-point, etc., could be made out to the observer, the patient stated that the feeling to him was 'different' over the areas described as numbed, saying that the sensation was 'duller.' His talking was a little thick and he stated that the tip of his tongue felt 'heavy'; sibilants were badly

pronounced. No paresis of the tongue muscles could be made out, and no alteration in the sense of taste. The mouth was sore and appeared inflamed; there were ulcers at the angles of the mouth and a few aphthae on the mucous membrane of the lips. The eyes showed superficial abraded areas on the lids at the margins, and there was a purulent discharge from the conjunctiva. Smears from the eye pus showed nothing particular, no inclusions detected, and on culture merely the usual organisms grew, mainly staphylococcus albus and micrococcus catarrhalis. In smears from the mouth and its ulcers no inclusions were seen, and on cultivation staphylococci, streptococci, etc., developed, nothing distinctive Faeces contained no helminth ova, and though constipated, appeared otherwise normal. Urine normal.

When seen again on April 12th he was much worse. I have in my notes on that day :—'He talks badly because, as he puts it, his "tongue is heavy and numbed"; there is still, however, no paresis; all the movements are carried out normally, and there is no atrophy. He only takes soup and soft food, he lies in bed without moving, but can move when he wishes. He states that he has "tingling in the face now."'

On April 15th when moving about in bed he fell off, and as he was restless a bed was made up for him on the floor. On the 18th profuse diarrhoea came on, 15 to 20 actions daily, and death occurred on the 20th. The mind was apparently quite clear till the last. Cerebro-spinal fluid had been taken, but examination of smears revealed nothing abnormal, the cellular elements were not increased, the fluid was clear and flowed drop by drop, and was not under pressure. The fluid gave a negative Wassermann reaction.

Blood examinations were made on March 28th and on April 12th. A few of the red cells showed some stippling, and others showed polychromasia, but these were few; no nucleated red corpuscles were seen, nor any parasites. No inclusions of any sort in the leucocytes. Differential counts of the latter gave the following results on the 28th of March and the 12th of April respectively :—

			Mar. 28th		Apr. 12th	
Polymorphonuclears			47·8		67·0	per cent.
Promyelocytes			0·2		0·2	,,
Myelocytes			0·2		0·8	,,
Metamyelocytes			0·8		—	,,
Eosinophiles			1·4		2·0	,,
Basophiles			0·4		1·0	,,
Large Mononuclears			3·2		3·4	,,
Transitionals			3·6		1·2	,,
Lymphocytes : large		9·6		4·6		
small		28·8		17·8		
Türk's		2·6		2·0		
Rieder		1·4		—		
			42·4		24·4	,,

Arneth count :—

	I	II	III	IV	V	S
Mar. 28th	5·4	38·5	35·6	12·9	5·5	2·1
Apr. 12th	7·7	43·0	33·4	9·6	4·8	1·5

Arneth Index :—

March 28th 61·7; April 12th 67·4.

It will thus be noticed here again how the differential count shortly before death approached the normal percentages, and the actual figures correspond remarkably closely with those of the last case, J.P.

At the post-mortem examination on this patient the following conditions were found macroscopically :—

Body emaciated ; no sign of any rash, either pellagrous or of any other condition. The loss of subcutaneous fat in this case also was very marked, the muscles were small but of good colour. Before undertaking any dissection of the body a lumbar puncture was performed. The fluid was clear and flowed readily, but not under increased pressure. Smears were made but revealed nothing abnormal, and cultural attempts on various media proved sterile as before.

Nervous system :—

Spinal cord. No sign of any inflammation of the meninges, and no haemorrhages The veins were congested, especially over the dorsal and upper part of the lumbar regions. The posterior part of the cord appeared softer than normal on section.

Brain. No sign of meningitis present ; the vessels of the surface were somewhat congested but not markedly so; the ventricles did not contain any excess of fluid.

Thorax. No pleural adhesions, and no fluid in the pleural cavities. The trachea contained a little yellowish, frothy mucus. The veins below the epiglottis appeared prominent. The tongue was covered with white fur which was easily removed ; there was no atrophy of the tongue or wrinkling of the surface.

Lungs. Left upper lobe was of a brick-red colour and congested, but crepitant all over and floated readily in water ; the right showed a similar congestion and coloration in the upper and part of the lower lobes, the middle did not appear to be affected. There were no signs of any tuberculosis.

Heart. Except for slight atheromatous patches at the root of the aorta, nothing abnormal was detected. The valves were all competent.

Abdomen —

Liver. This organ showed puckered areas over the right lobe, and on section the tissue appeared somewhat darker than normal and showed a similar cicatrix ; nothing resembling ordinary gummata.

Spleen. Capsule thickened ; tissue dark but of normal consistence ; organ not enlarged.

Kidneys. Apparently normal.

Stomach. Contained about half a pint of greenish watery fluid and grumous mucoid material of the same colour ; the mucous membrane was to all appearances normal.

Small Intestine. Mucous membrane normal till the lower three or four feet were reached, when the vessels became prominent and increasingly so down to the ileo-caecal valve, when the congestion became acute. Scattered at intervals were some half a dozen dark, pigmented patches, visible also from the serous surface. Peyer's patches were not unduly prominent ; solitary follicles clearly defined.

Large Intestine. Mucous lining pink, but congestion by no means marked.

Mesenteric glands. Slightly enlarged.

The results of the microscopical examination will be detailed after the other cases have been described. The same tissues were taken as from the last patient.

(*c*) T.J., male, aged 28 years. Both the onset of the symptoms and the course of the disease were slower in this patient than in any of those previously recorded. The history given by this patient was that until March 1st he had been quite well. On that date his right eye began suddenly to itch and became red ; two days later the left was similarly attacked ; on the 15th (a considerably longer interval than in the last case) the mouth became affected ; it felt sore and hot

so that he was unable to take his food for 24 hours. After this he could eat without pain. His feet began to feel numbed on the 20th or 21st, and this condition spread upwards so that on presenting himself at the hospital on the 23rd, though he was able to walk, he had not complete control over his legs. He had been very constipated from the onset of his illness.

Examined by me on March 28th when the following notes were made :— 'Eyes not very inflamed now, but a viscid purulent discharge is present, and there are shallow abrasions at the edges of the lids and on the palpebral conjunctiva. The mouth is sore ; there are ulcers at the angles and shallow aphthae on the mucous membrane of the lips. The tongue is not affected. He is able to walk, but, when getting out of bed, the legs are thrown about ; the gait is ataxic but not stamping ; he can turn without stumbling ; knee-jerks are absent ; to the Babinski test there is no response. He states that there is "numbness up to the knees," but objectively no alteration of sensation can be detected, though the patient states that "touch feels different" over the area from the knees downwards. As far as can be tested Romberg's sign is not present. Pupils react normally both to light and convergence. The faeces contain ova of ankylostoma and ascaris.'

He was examined again on April 12th when his condition was found to be considerably worse. He was unable to walk, and when supported on both sides in attempts to walk, the legs were quite uncontrollable and were thrown about in all directions, sometimes spread wide, at other times catching in each other. 'He complains now (April 12th) of tingling and numbness in the fingers and of "pain" in the upper arms; no pain at all in the joints. Both hands were affected together and the sensation is spreading upwards daily. The tongue feels numb at the tip and his speech is altering, owing, as he states, to a "heavy feeling in the tongue," and there is obvious difficulty in pronunciation of certain words, especially those containing sibilants. He is quite rational and answers all questions readily, promptly and intelligently.'

When seen again on April 25th he was totally unable to walk, even when supported by an attendant on each side. 'Talking is difficult, so he rarely speaks, but when addressed he answers rationally as before. He is clearly going from bad to worse.' Position and joint senses considerably affected, apparently, but his dysarthria makes accurate estimation very difficult. Cerebro-spinal fluid was taken on this occasion ; it flowed readily, but was quite clear and limpid ; nothing abnormal was detected in smears made of the fluid as it flowed, and also after prolonged centrifugalisation ; there was no deposit. Cultural attempts all remained sterile, and the Wassermann reaction carried out with this fluid and with the blood was negative in each case.

The patient remained in this condition for nearly three months without any further spread of the numbness and, in fact, without development of any fresh symptoms except that he was becoming gradually weaker and weaker. During this time the bowels were opened naturally about once a day, occasionally twice and sometimes only on alternate days ; the stools were natural. On July 19th, however, diarrhoea began to set in, five or six actions occurring daily, increasing to ten, and the patient died three days later. When seen on the 19th, though he did not not move voluntarily, but lay quiet in bed, there was no real paralysis detected ; movements of the limbs, though weak, were all carried out normally ; tongue not affected as regards its movements.

Blood examination was carried out on April 12th and again on July 19th.

I regret to say that I cannot find the records of the total counts, but the differential leucocyte counts on these dates were as follows :—

		April 12th		July 19th	
Polymorphonuclears		49·8		54·6	per cent.
Promyelocytes		0·2		0·8	,,
Myelocytes		0·4		0·6	,,
Metamyelocytes		0·8		1·0	,,
Eosinophiles		3·0		4·8	,,
Basophiles		1·2		1·4	,,
Large Mononuclears		2·0		2·4	,,
Transitionals		2·0		2·2	,,
Lymphocytes : large	6·2		5·0		
small	32·2		25·0		
Türk's ...	1·8		1·2		
Rieder type	0·4		1·0		
	——	40·6	——	32·2	,,

Arneth counts :—

	I	II	III	IV	V	S
April 12th	8·4	53·4	24·6	8·4	2·4	2·8
July 19th	8·7	54·6	23·9	8·1	1·8	2·9

Arneth Index :—

April 12th 74·1 ; July 19th 75·2.

(*d*) M.H., female, aged 30 years. This case ran a more prolonged course than the first one recorded in this series, but less than the last, the total duration being about two months. The history of this patient is somewhat vague ; her statements as to the chronological order of the events were not altogether consistent. At one time she stated that the numbness was the first symptom, but patient enquiry elicited that the course was probably as follows :—

On or about March 12th her eyes and mouth became 'sore and itched' ; she could not remember which was affected first ; eight days later she felt a 'tightness of the chest' or, as she sometimes described it, 'a rope feeling' there. At the end of the month (28th or 29th) her toes and the soles of her feet felt numbed, and this sensation extended to the knees and interfered more and more with her walking.

When seen by me on April 12th she was totally unable to walk and could not even get out of bed. The knee-jerks were exaggerated and multiple, patellar and ankle clonus were marked. This had been the only case so far of those recorded in which the reflexes were increased. The sensory alterations were subjective only, as in the other patients. In the upper limbs the reflexes were normal. Pupil reactions normal ; no inflammation of the eyes or mouth now, no affection of speech, and no tremor, either volitional or when at rest.

Her blood was taken on this date and gave a negative Wassermann reaction ; the blood count was very similar to that of G.P. [No. (*b*) of this series] towards the end of his illness, except that the percentage of large mononuclears and transitionals is higher ; the differential leucocyte count gave the following results :—

Polymorphonuclears	...	...	...	...	60·4 per cent.
Promyelocytes	...	...	...	...	0·2 ,,
Myelocytes	...	...	...	...	0·6 ,,
Metamyelocytes	...	...	...	...	0·2 ,,
Eosinophiles	...	...	...	...	3·2 ,,
Basophiles	...	...	...	...	1·2 ,,
Large Mononuclears	...	...	...	...	5·8 ,,
Transitionals	...	...	...	...	3·0 ,,
Lymphocytes: large	...	...	...	6·6	
small	...	...	...	17·8	
Türk's	...	...	...	0·4	
Rieder type	...	...		0·6	
				——	25·4 ,,

Arneth count:—

I	II	III	IV	V	S
8·9	46·4	27·5	11·3	3·3	2·6

Arneth Index 69·0.

Her further history showed that the symptoms took on the character of the cases already described; thus, the reflexes soon disappeared, the condition of numbness extended to involve the hands, and later the tongue. Her temperature on admission to hospital was 100° F., but thereafter, with one exception when 99° F was recorded, was normal. She suffered from constipation until the 7th of May when the actions became looser, and diarrhoea set in, and the case terminated fatally on May 14th.

(*e*) J.M., male, aged 28 years; the husband of the patient, E.M., whose case has already been described. J.M. was admitted to hospital on April 19th when the history obtained from him was that towards the latter part of February his eyes became sore and inflamed, 'they itched and were red.' Some time later (he could not state definitely how long was the interval) his mouth also became sore. After this no further symptoms declared themselves, except for troublesome constipation, until March 28th when he felt a 'numbness' in the feet—the toes and soles—which in the course of a few days extended to the level of the knees. He never felt any 'fever.'

When seen on April 12th his lids showed abraded areas, and there was a mucopurulent secretion present; the mouth was ulcerated at the angles and fissured; the mucous membrane of the lips was inflamed, but he was able to take food without pain. He was unable to walk without help; when supported he had an ataxic gait, and suggestive of spasm, but not tabetic; he was not able to stand alone, so Romberg's sign could not be tested. Tactile sense appeared normal with the usual tests, though the patient himself stated that he could perceive the difference over the areas complained of as being numb. Sensation of heat and cold, joint, and vibration senses apparently normal. Knee-jerks absent. Allowing as far as possible for the ataxia there was no detectable loss of power in the leg movements. Hand and arm movements normal; grip good.

On April 25th the blood was taken for a Wassermann reaction and the cerebro-spinal fluid for the same test; smears and cultures were also made with the latter. Nothing abnormal was detected, and the culture tubes remained sterile. The Wassermann reaction was quite negative with both the blood and the cerebro-spinal fluid. I have the following note on that day (April 25th).—'Patient about

the same, no improvement, but no further extension. The numbness reaches just to the thighs, but neither the hands nor the tongue are affected at all. He is no longer constipated, but the bowels are acting normally, as a rule once daily, occasionally twice.'

His condition remained practically unchanged for nearly four weeks after this, when on May 21st diarrhoea set in. There was no further spread of the nervous symptoms except that defective estimation of joint and position senses ensued; his replies to questions as to relative weights were too variable for certain record. He appeared to guess rather than to reply intelligently, but there was almost certainly some defect in the power of estimating weight differences. The number of actions of the bowels on the 22nd, 23rd, 24th and 25th, was six, four, six and fourteen respectively. The temperature which had been normal ever since his admission to hospital, rose slightly to 99·2° F. on the 26th May, on which day the patient died.

I am unable to find my notes of the blood examination of this patient, except the record of the Wassermann reaction.

It is worthy of note in this case that he suffered from constipation from the onset and while the nervous symptoms were developing; that when these latter came to a standstill the bowels acted normally, and towards the end, as in the other cases, diarrhoea either led to death from exhaustion or at least was closely associated with the fatal issue.

8. Lastly a brief description will be given of two cases which did not arise at Spanish Town, but which were sent to me by a practitioner in Kingston who had heard of the symptoms presented by the patients at the former place. His remark in sending them was to the effect that the patients were examples of what had been called 'peripheral neuritis' in Jamaica for many years, and he added in each case: 'Is this anything like those seen in the Spanish Town epidemic?'

The first one closely resembles the condition already noted, except that the course was much slower and the knee-jerks were exaggerated; the second differs in that the knee-jerks were present and the disease, so far as the patient could recollect, was not ushered in by eye or mouth symptoms. Thus they correspond somewhat with the case M.H., described above, in which at one stage a similar state of the reflexes was found, though later on they were lost. The patients left for the country parts the day after my examination, and I have not been able to hear anything about either of them since. Possibly in their cases, as time went on, the same disappearance of reflexes may have occurred, but this is uncertain.

(*a*) J.N., male, aged 24 years. This patient stated that in December 1916 his eyes 'became sore'; there was no photophobia, but there was a slight discharge and the lids were gummed together in the morning on waking. Three weeks later the mouth became affected, being sore particularly at the corners; the condition, however, never interfered with his taking food. One week subsequent to this, or four weeks after the onset, the toes and soles of the feet felt numb. This numbness gradually extended over the dorsum of each foot and up the legs; when seen on April 26th it extended above the hips. Early in February the finger-tips felt numbed, and by the time that I saw him (April 26th) the backs of the hands were similarly affected. There was no numbness of the tongue.

When examined on the 26th April the gait was suggestive of a spastic condition, but not markedly so, rather of a steppage type. The patient stated that the ground felt 'springy' and as if he were "walking on indiarubber." The knee-jerks were exaggerated, and there was a suggestion of ankle-clonus; Babinski's

test gave possibly a slight extensor response, but the skin of the soles was very hard and thick so this point could not be determined satisfactorily. No Romberg's sign present, no nystagmus, no Argyll-Robertson pupil; no loss of power detected. No sensory defects made out objectively, no affection of sphincters. Patient had suffered with constipation throughout.

Blood examination on April 26th gave the following results :—

Erythrocytes 4,400,000 per c.mm.; Haemoglobin 76 per cent.; Colour Index 0·86. No nucleated red cells seen, no malarial parasites found, no poikilocytosis, but some degree of anisocytosis.

Leucocytes 8,700 per c.mm.; differential count gave :—

Polymorphonuclears			41·6 per cent.
Promyelocytes			0·6 ,,
Myelocytes			1·8 ,,
Metamyelocytes			0·4 ,,
Eosinophiles			1·6 ,,
Basophiles			2·0 ,,
Large Mononuclears			4·4 ,,
Transitionals			2·2 ,,
Lymphocytes : large		10·8	
small		32·8	
Türk's		1·0	
Rieder type		0·8	
		——	45·4 ,,

Arneth count :—

I	II	III	IV	V	S
6·3	35·1	32·7	14·0	6·3	5·6

Arneth Index 57·7.

The Wassermann reaction carried out with the serum from this patient was negative.

(*b*) S.C.T., male, aged 36 years. This patient states that his previous health has always been good with the exception of an attack of 'lumbago' in 1912. He cannot say how long the attack lasted, but knows that it 'quite cleared up in a comparatively short time.' Since then he has remained perfectly well until December 1916, when he first began to feel a numbness in the toes and fingers. 'He thinks that the fingers were first affected, but is not sure. The change of sensation at the beginning was very slight indeed, and the feet and hands were affected so closely together that he does not remember the order of involvement.' Since that time walking had become increasingly difficult, so that when I saw him on April 3rd he dared not venture without the aid of a stick, and he stated that he found that at home he walked better and more steadily when holding a chair. When demonstrating this to me he held the chair in front of him off the ground, not in any way as a support, but, as he put it, 'to balance himself.' His complaint at that date was not of numbness but of 'weakness' in the legs and hands. The weakness, however, must have been very slight, for the chair which he used was a comparatively heavy one of mahogany, and he held it out by the top rail in front of him, as already stated. There had never been any tingling or pricking sensation.

At the examination of this patient on April 3rd the following notes were made in addition to the points given above :—

'No apparent affection of the auditory nerves, reacted normally to all the usual tests, watch, tuning-fork, etc., no nystagmus, no diminution of the field of vision, no ocular paresis. Romberg's sign not present; he stands quite steadily with eyes closed, but states that when washing his face he usually steadies himself with one hand on the wash-stand. Knee-jerks present, possibly a little exaggerated, but not brisk or multiple, and there is neither patellar nor ankle clonus. Babinski doubtful, skin thick. Gait is suggestive of spasm, but very slightly. No affection of sphincters. Touch is plainly felt and accurately located everywhere. Joint and vibration senses normal, also sense of position. Heat and cold less certain, six times out of ten he described the hot tube as cold. There is no loss of power in any of the movements beyond what might be ascribed to his not using his leg muscles much of late. The legs are thin; but muscles are not flabby, and electrical reactions are normal. Measurements on the two sides are equal. No tremor of fingers or lips, and there is no affection of speech. The grasp is firm and strong, although he complains of "weakness" in his hands. He says that the ground does not feel soft or woolly, but that he has a sensation as if the floor were "slipping from under him." There is no pain anywhere but a feeling of "weakness" in the knees. In spite of the numbness or weakness of his fingers he unlaced and laced up his boots fairly rapidly and without any difficulty or fumbling. He denies all venereal disease, and there is no evidence of any such.'

The Wassermann reaction was quite negative. A differential leucocyte count gave the following results:—

Polymorphonuclears			37·6 per cent.
Promyelocytes			0·8 ,,
Myelocytes			0·4 ,,
Metamyelocytes			0·2 ,,
Eosinophile Myelocytes			0·2 ,,
Eosinophiles			10·0 ,,
Basophiles			2·2 ,,
Large Mononuclears			3·0 ,,
Transitionals			4·2 ,,
Lymphocytes: large	12·0		
small	26·6		
Türk's	1·8		
Rieder type	1·0		
		41·4	,,

Arneth count:—

I	II	III	IV	V	S
2·7	26·1	34·0	25·0	5·3	6·9

Arneth Index 45·8.

No malarial parasites were seen; no specimen of faeces was obtainable for examination for ova of ankylostome or other worms to account for the eosinophilia. They are present in so large a percentage of natives here that it is more than probable that these cells may be thus accounted for. Far higher numbers may be met with in patients harbouring these parasites here (I recently saw one with 37 per cent.), although there are no obvious indications of helminthiasis in the general state of health, no anaemia, no oedema, shortness of breath, and so forth.

IV. SUMMARY OF EXAMINATION OF THE BLOOD

The Wassermann reaction was carried out with the blood of ten of these patients, and in several instances with the cerebro-spinal fluid also, but in every case with negative results. This was done for two reasons: firstly, because it was a natural surmise that patients showing these peculiar nervous symptoms might be syphilitic; secondly, because the suggestion has been made that so-called peripheral neuritis, which is comparatively common here and which so markedly resembles the chronic stage of the condition under consideration, may be sequel to yaws or a 'paraframboesial' affection analogous to tabes dorsalis as a form of parasyphilis.

The results of the Wassermann tests may be summed up in the one word 'negative.' In no instance was a positive result obtained with either the blood-serum or the cerebro-spinal fluid. The tests were performed with every care; standardization is made of the haemolysin, the complement, and the antigen in this laboratory every week when Wassermann reactions are carried out, and the above sera and fluids were tested at the time the routine tests were done, from patients in the hospital and from outside, so that there were abundant controls.

The uniformly negative results dispose of the idea that syphilis is at the root of this peculiar disease.

Passing on to the other blood examinations, it will be seen that total blood-counts were made on eight occasions and differential leucocyte estimations on twenty-eight.

Dealing first with the erythrocyte counts and the haemoglobin percentages, the results in all the cases bore a remarkable resemblance to each other. Thus, in D. J., a mild case of the intestinal form with recovery, there was a 22 per cent. reduction of red cells and a 30 per cent. reduction of haemoglobin, giving a colour index of 0·89; comparing this with E. G., a severe and chronic case of the nervous form, at the stage prior to the acme, there was a reduction of red cells of 34 per cent. and of haemoglobin 40 per cent., giving a colour index of 0·9; at a later examination of this last case, at the height of the disease, when the nervous symptoms were at their worst but the general condition was fair, there was a reduction of 31 per cent. in the red cells and a 40 per cent. in the haemoglobin,

giving a colour index of 0·89; while yet again, when the condition had become chronic, but the general health was still maintained, there had been a little improvement in the erythrocyte count, the reduction being 28 per cent., and the haemoglobin had improved 4 per cent., so that the colour index was 0·88. The difference between these is so slight as to be almost negligible. The condition found in a severe and chronic case, which, however, terminated fatally, showed that there was a gradual reduction in both red cells and in haemoglobin, but not very marked; in fact, when this patient was at the last stages the findings were very nearly identical with those just given in the case of E. G., where the condition was becoming chronic and the general health was fair.

In this case of J. P., when he was in an early stage, up and about, the red cells were 4,200,000 per c.mm., and the haemoglobin 80 per cent., giving a colour index of 0·95; when he was bedridden, and the symptoms were widespread, there had been a further reduction of corpuscles of only 8 per cent. and similarly of haemoglobin, so that the colour index remained the same. Lastly, shortly before death, a third estimation showed only a 4 per cent. further reduction of red cells, with haemoglobin 62 per cent., giving a colour index of 0·86.

Finally, in the case of J. N. (if this be regarded as belonging to the same category, but of a very chronic course), when his general health was good and he was up and about, the colour index was the same as that in the last patient when in extremis, the red cells being 4,400,000 and the haemoglobin 76 per cent.

Practically the only inference one can draw from the above is that the blood condition, as regards the erythrocytes and their haemoglobin content, depends rather upon the general state of health than upon the degree and extent of the symptoms accompanying it.

Comparing the total leucocyte counts in the same patients, it will be seen that the average departs very little from the normal. The mild case of D. J. had 8,400 leucocytes per c.mm.; the very chronic case in a good state of general health, J. N., 8,700. In the severe chronic case, E. G., there was a slight reduction from 9,200 in the earlier stage to 9,000 at the height of the affection, and an increase to 10,100 when the chronic state was fully established, but the general health was fair.

Reviewing the findings in the severe and fatal case of J. P., we see that at an early stage the leucocytes numbered 8,600 per c.mm.; when he was much worse, bedridden and helpless, the leucocytes had increased by 1,000 per c.mm., while the red cells had increased somewhat, and again at the termination of his illness, they had further risen to 11,200, which was only 1,100 more than E. G. when in fair health. The total counts are, therefore, not definite enough to warrant any conclusions as to the cause of the disease or its mode of action, nor do they afford any reliable indications on which to base a prognosis or estimate the stage of the disease.

Passing on to the differential leucocyte count: the accompanying table (Table I) gives the detailed results in percentages. For the estimation of the leucocytes in no case was less than five hundred counted, and in some instances seven hundred to one thousand; while for the Arneth count also at least five hundred were noted. This, as will be readily understood, was a laborious and somewhat tedious undertaking, all the more disappointing in view of the barrenness of inferences to be drawn from it. Each patient appeared to be, as it were, a law unto himself. No sooner did one seem to observe changes in a definite direction in the course of a case, such as, for example, an increase in polymorphonuclears and diminution in the high lymphocytosis, than another analogous case would be met with where none of these changes could be found.

As regards the polymorphonuclears, it is perhaps worthy of remark that the only cases in which the relative proportion of these cells approached the normal, namely Nos. 21 and 23, in the table, were in fatal cases towards the end where the patients were practically in extremis. On the other hand, those who showed the greatest departures from the normal, namely No. 7 with only 27·2 per cent., No. 13 with 30·6 per cent., No. 9 with 33·8 per cent., and so forth, were patients who were not seriously ill or who were recovering.

At the same time it must not be forgotten that in every instance there was found a small proportion of immature forms—promyelocytes, myelocytes and metamyelocytes.

Eosinophilia only occurred in patients whose faeces contained ankylostomes, either alone or with other helminth ova (but see remark on the patient S. C. T., page 142), and in all who had eosinophile

myelocytes, ova were present. One patient, A. D. (No. 18), was harbouring both ankylostomes and ascaris, and yet showed no increase in eosinophiles. A slight relative basophilia was noticed in the majority of cases. The significance of this is doubtful, but it is interesting to note that this condition has been recorded in patients convalescing from beri-beri. Basophilia, however, is not an uncommon condition here; I do not know whether it has been noted in other tropical countries, but I have counted as high a proportion as 5 and even 8 per cent. in the blood of a person in apparently perfect health.

The large mononuclears and transitional cells were increased in a few patients only, and in them not to a great degree. The highest found was 11·2 per cent. in A. B., and she also showed malarial parasites in her blood; with this exception, the highest reached 8·8 per cent. (No. 26) at the height of her illness, which terminated fatally. The average of all those recorded amounts only to 5·9 per cent., or, if the malarial ones be excluded, to 5·7 per cent., in other words, within the limits of the normal. The lymphocytes were increased relatively in all cases except those already mentioned when speaking of the polymorphonuclears, in which the patients towards the termination of their illness showed a differential count closely approaching the normal. Not only was there this lymphocytosis present in all the others, but also in every case there were present the abnormal forms (abnormal, that is, in their entrance into the general circulation) of the Türk's irritation cell and the Rieder type.

Lastly, considering the results of the Arneth counts: Here again we can find nothing on which to base any inference. Two years ago I made a large number of Arneth estimations on healthy subjects, both European and native, and found that compared with what has come to be regarded as normal in England the count in this part of the tropics tends to shift to the left. When I was getting my results together for publication, two papers appeared in the *Annals of Tropical Medicine*, by Scott Macfie (1915), and by Breinl and Priestley (1915), which covered the same ground, and in them the results given were so similar that I saw no object in making my own findings more widely known.

The question of the Arneth count in the cases of the disease

under discussion may be summed up briefly thus: Though the Arneth index is generally higher than that recorded in Europeans at home (about 40), it shows very little difference from what has been found in the blood of healthy Europeans in the tropics (51), and no difference at all as compared with apparently healthy natives (55·9).

The highest index in all of the series given in the table was 69·4, a case of the nervous variety of the disease in an advanced and chronic stage, and the lowest (if we except the uncertain case, No. 28) was in the early stages of a severe and fatal case of the same (No. 19), in which the index was 38·3. The average for the whole series works out at an index of 51·2, or if we exclude the two uncertain cases (27 and 28), at 52·4, or approximately that found in healthy natives.

No inference, therefore, is permissible, unless it be that the condition is probably not a blood infection.

A relative lymphocytosis is well known in the tropics; not only in association with intestinal conditions, such as some of these patients exhibited, but so frequent as to be regarded as almost the normal state.

In the counts given in the table, the lymphocytosis is in excess of what can be looked upon as a normal condition, or even as one frequently encountered. The condition presented by these cases, however, does not correspond with any of those generally noted as associated with an absolute or a relative lymphocytosis, with the exceptions of syphilitic affections and intestinal diseases. The former of these is excluded by the uniformly negative Wassermann reaction. How far the latter fulfils the possibilities will be discussed in the sequel.

V. SUMMARY OF NERVOUS SYMPTOMS

Table II gives a general summary of the chief symptoms exhibited by the various patients whose histories and physical conditions have already been detailed. A perusal of this table will obviate a long description of the various features presented.

In all cases except one the intellect is stated as good, and it remained so till the end. In the one exception, one might with

fairness state that the fault lay less in the general intellectual condition of the patient than in her memory for details. She replied promptly enough to questions and evidently understood them clearly, but owing to a defective memory her replies were at times contradictory when the questions related to previous events, in particular the onset of her illness. In dealing with the symptoms as set down in the table, it will be better to exclude the last two cases. These were only seen on one occasion, they had been ill for a long period, and it is far from certain whether at all they afford examples of the condition under discussion. They were sent because some of the symptoms presented resembled those found in the Spanish Town outbreak, and the medical practitioner who kindly sent them to me stated that he did so on account of this resemblance; since, however, only prolonged observation could decide the question and the patients had to leave Kingston for the country the same day, no assertion as to the identity of their condition with that under review can be made.

With regard to the remaining nineteen patients the onset occurred in every case comparatively suddenly. The first symptoms were the same in almost every instance—sore eyes and mouth. All suffered with one or other, or both. One stated that her mouth was affected as the first symptom, and that there had been no eye trouble at all, another was a little doubtful on the point, while two stated that the itching and soreness of the eyes had been succeeded in course of time by the nervous symptoms without any intervening mouth involvement.

It will be also seen that the 'intestinal' or diarrhoeal cases never showed any nervous system affection at all, and that none of the 'nervous' cases had any early diarrhoea; all were constipated. This fact has already been noted in an earlier section, and need not be laboured further now.

Passing on to a brief consideration of the nerve affections: All complained of a numbness of the feet and legs. In more than half, this sensation extended above the knees, and in all but one of these in whom this extension took place the upper extremities became involved later, while in four cases the tongue was affected also. In no patient did the latter occur without involvement previously of the hands.

In five instances the girdle sensation was complained of; but in none of these was any zone of hyperaesthesia detected at or above the level of girdle sensation.

In no case was any real 'pain' given as a symptom. Three of the patients mentioned pain; one stated that it was in the knees, but only one movement, and it appeared to be slight; there was no heat or swelling and no pain elicited on passive movement, manipulation of the joints being allowed without resistance. Another stated that she had pains in her arms at night, but, on further questioning, as already stated (case of C. H.), she explained that she did not feel any real pain, but that the arms felt uncomfortable and that she could not place them in a position of ease during the night. A third (T. J.) stated that with the tingling and numbness of the hands he felt 'pains' in the arms, but here, too, movement and manipulation were permitted freely, so that it cannot have been at all severe.

Three only stated that they had any 'crawling' sensations, and tingling was not very common; the noteworthy point about all the cases was that in spite of the extensive area over which they felt the numbness, no change in ordinary tactile sensation could be made out objectively, except in one patient (E. G.), where there appeared to be a slight recognizable deficiency. As stated in the preliminary remarks, all the patients were able to feel the slightest touch when their eyes were bandaged, and furthermore could accurately locate the part touched. In severe cases there appeared to be some affection of the kinaesthetic sense, and weight discrimination was markedly deficient in two. In many, I am sorry to say, this test was not made.

Although no interference with tactile sense could be detected objectively, nevertheless some of the patients affirmed that over the numb areas, although they could feel light touches, the sensation conveyed was 'duller than' or 'not the same' as over the normal parts.

The only sensory change which could be definitely made out objectively was that of discrimination between heat and cold. Even where the differences were great, e.g. cold water in one tube and water recently boiled in the other, confusion often resulted, and, strange to say, the reply was more often to misname the hot tube

and say it felt cold than *vice versâ*, and again in many instances where the discrimination was made the sensation was distinctly delayed. The hot tube could be applied for three or four seconds before the patient recognized it as hot, and even then was at times dubious.

In three cases in which the hands and forearms were affected, astereognosis was present, and in one (E. G.) was very marked, as has been noted in the detailed report.

There was no localised wasting of muscles. As the disease progressed to a fatal issue a condition of general emaciation came on, but no true paralysis of any group of muscles could be detected. The movements generally towards the end were weak, but this was merely the result of the exhaustion and not due to paralytic conditions.

The gait was variable, at times inclined to a spastic condition, the feet, as it were, sticking to the ground and shuffling; in many it was ataxic, the feet being thrown about wildly; in none was it really tabetic; in some, again, it was a 'delicate' half-shuffling, half-stumbling gait, while in many it could not be tested owing to the patient being unable even to stand, much less walk Romberg's sign was doubtful in two cases, negative in all the rest in whom the test was possible.

With regard to incoordination: All those in whom the numbness had extended to the knees showed incoordination of the lower limbs, but in the majority this was much less marked when the patient was lying in bed than when he was up. Those patients whose hands or arms were affected showed a corresponding incoordination of the hands.

Pupillary reactions were normal in all cases except in one who had a somewhat sluggish reaction to convergence; in no instance was the Argyll-Robertson phenomenon present. The field of vision by ordinary rough tests was normal, and there was no instance of strabismus, ocular paresis, or diplopia. In those who were passing to the chronic stage with recovery of general health, a frequent complaint was of 'dark vision.' The significance of this term has been explained in the section giving the details of the various cases. In some of these chronic cases, in patients recovering general health with residual symptoms, as already mentioned, some degree of

deafness was found. This showed itself as a defective nerve conduction.

Taste was normal in all; the testing was performed in the usual way with sugar, salt, quinine, and citric acid. Even in those who complained of the numbness of the tongue no defect in taste could be detected. The reflexes depended apparently to a great extent upon the area affected by the numb sensation. In no case was numbness of the face complained of, though one patient (A. D.) stated that her 'face felt tight, but not numb.' The jaw-jerk was normal in all. The elbow-jerk was doubtful in two patients, and absent in E. G. The wrist-jerk was absent in all those in whom the numbness extended to the forearms. The knee-jerks were lost early in the affection, at all events by the time the patients came under observation. In most cases this was not until the numbness had spread to the knees and made walking very difficult or impossible. The possibility of there being an initial exaggeration must be considered in the light of the patient M. H. In her the onset was slow and irregular, and the knee-jerks at an early stage were exaggerated, but later, as the disease progressed, they disappeared, and the same remarks apply to the ankle-jerk.

Babinski's test is quite unreliable in the native. The skin of the sole is so thick that in 90 per cent. or more no response at all is obtained. The superficial reflexes were in nearly all cases normal, in three they were noted as variable, by which is implied that they were indistinct and obtained irregularly.

In no case was there any sphincter affection. Even in the bed-ridden patients there was never any need for a catheter, and, as stated in the reports on, for example, J. P. and G. P., when the profuse diarrhoea came on at the termination of their illness, there was not the involuntary evacuation of sphincter involvement, but the stools were merely loose and frequent; the patient was always able to inform the attendant of his wants.

VI. HISTOLOGICAL CHANGES

The changes in the nervous system are very marked and widespread, and, as will be inferred from the description of the symptoms present, there is no picking out of any one system or tract corresponding, for example, with anterior poliomyelitis.

Since the nervous system changes are the most marked and most important, they call for more detailed description than the other organs of the body. The conditions found in the latter will be dealt with first and more briefly.

Parts of various muscles were taken, viz., heart, supinator longus, tibialis anticus, tongue. The skeletal muscles showed the changes about to be described.

Tibialis anticus: The transverse striation of the fibres is well marked and the nuclei stain well; here and there, but by no means frequent, one sees a fibre which has poorly-marked striation and in which the tissue appears granular; in such the nuclei stain less well. This condition is shown by the ordinary staining methods—haematoxylin and eosin, haematoxylin and Hansen's modification of van Gieson, etc.

The tissues which have been subjected to the Marchi method or other osmic acid process show, in a fair proportion of the fibres, small black particles which under higher magnification were seen to be globular—fat. These fibres show the transverse striation, but not as plainly as in normal muscle; other fibres appear vacuolated and may contain comparatively large fat-droplets. Fibres showing well-marked striation contain no granules. Affected fibres are apparently picked out arbitrarily, healthy and granular fatty fibres lying side by side.

Supinator longus: Similar to above, but in less degree. No fibres seen showing large fat globules; comparatively few show even the small granules, and the number of fibres normal is much greater in proportion. Even those showing most granules have not completely lost their striation. By haematoxylin and Hansen nothing abnormal seen, except that a few of the fibres show some excess of nuclei.

Tongue: No atrophy of muscle, epithelium normal, muscle nuclei stain well, striation well marked. No abnormality seen.

Heart muscle: The cross-striation is variable in distinctness, in some parts quite plain, in others it is partly obscured. Here and there are seen small patchy accumulations of round cells in the sub-epicardial layer. The longitudinal striation shows plainly.

In the corresponding tissue from another patient the changes are

similar, but in addition there is a slight interstitial myocarditis, with dilatation and congestion of the cardiac capillaries. The muscle-nuclei as a whole stain well. By the Marchi-Alghieri method, minute granules and droplets stained black by the osmic acid are fairly generally distributed throughout the muscle tissue. Occasionally one sees a fibre showing no proper striation, and here the granules are massed together, in some situations aggregating to form minute droplets. Some of the granules are of a browner colour, and may be pigment, not fat; in favour of this is the fact that many of the fibres showing numerous granules have well-marked striation and no signs of degeneration, but on the other hand sections stained with haematoxylin and van Gieson (Hansen's modification) which have therefore passed through alcohol and xylol, show no granules, thus pointing rather to their fatty nature.

Lung: Some degree of emphysema. Vessels are congested and the alveoli in isolated spots contain red blood cells. In other places there are localised areas of broncho-pneumonia, the alveoli containing red cells, leucocytes and shed epithelium, adjacent to distended emphysematous alveoli, and the bronchioles have also lost some of the lining epithelium.

Spleen: Haematoxylin and Hansen. In the one case the capsule is thickened; Malpighian corpuscles well marked; vessel walls thicker than normal, and fibrous trabeculae prominent. There are pigmentary deposits (brownish black) scattered irregularly throughout the pulp, not more numerous in the neighbourhood of the vessels. In the tissue from another patient there is no excess of fibrous tissue, nor thickening of the vessel walls; very little pigment seen.

In Marchi sections there is considerably more black staining than in the haematoxylin sections, pointing to the fact that in addition to the pigment noted above there is also some fatty metamorphosis or deposit, which latter had been removed in the preparation of the tissue for the haematoxylin but retained by that for the osmic method. Under the higher powers the brownish-black granular pigment can be distinguished from the globular, black fatty deposits.

Liver: Haematoxylin and Hansen. G. P. showed accumulation of small round cells and early fibrosis in places, especially round the portal system. Also subcapsular accumulations of similar cells, and

here and there beneath the capsule are spaces filled with blood corpuscles; the outer edge being limited by these small-celled accumulations. In one part of the liver the development of fibrous tissue is more marked and in its neighbourhood are spaces filled with red blood cells, the liver cells showing as mere strands, as it were, resembling an angeiomatous condition. Cell-nuclei stain well. The fibrous strands pass in from beneath the capsule and divide the tissue into lacunae containing blood, the liver cells in small masses looking like islets with thin connecting strands in a lake of blood. In the other case there is similar small-celled infiltration but in larger masses, apparently mostly in the region of the portal canals; the liver cells in such situations appear 'rarefied' and partially displaced. These parts resemble alveolar tissue with small cells and liver cells in the meshes. The line of demarcation between this condition and normal liver cells is moderately abrupt.

Kidney: Haematoxylin and Hansen. The nuclei in glomeruli stain well, but in some instances the whole glomerulus is shrunken from Bowman's capsule. There is slight increase of the interstitial tissue. The nuclei of the secreting epithelial cells stain well on the whole, but in places the protoplasm is granular and stains poorly, especially that of the convoluted tubules and the ascending tubules of the loops of Henle. In one situation there is a marked localised development of fibrous and unstriped muscle tissue, fairly abruptly demarcated from the surrounding renal tissue; there is no small-celled infiltration, no caseation, nuclei stain well.

Marchi. In tubules with degenerated epithelium minute black granules and droplets are seen, not marked in the convoluted tubules. The tubules so affected, however, are not relatively numerous, though by the 1/12in. very small dots may be seen in many in which they are not recognized by the lower powers. The cells of the glomeruli occasionally show the same condition, but the affection of these is very slight. Under the high magnification it is noticed that the majority of the tubules show very small black dots and granules. They may also be seen in the vessels, but not commonly, nor are the droplets at all large there.

In Marchi sections the glomeruli are seen not to be so shrunken from the membrane, and the condition present in the haematoxylin section is, therefore, probably an artefact in the preparation.

In the second patient, except for a slight increase of intertubular connective tissue, the appearances are nearer normal. The renal epithelium shows well-marked nuclei, though the cell-limits themselves are not well defined. The droplets and granules also are much less numerous than in the previous case.

Though many of the 'particles' are globular in shape and black, others appear more of a dark brown, amorphous, pigmentary deposit.

No organisms, bacterial or protozoal, seen.

Adrenals: Very little departure from the normal seen by the haematoxylin method, no pigment visible, and the nuclei stain well. In some parts the cells appear a little more granular, and some give a faint suggestion of vacuolization. By the osmic acid staining the majority of the cells show black granules, which in many instances have coalesced to form spherical droplets. The interstitial tissue shows a similar deposit of black granules, but here they are smaller and have not coalesced. The zona fasciculata seems to show the condition in most marked degree; the reticulata least.

Pancreas: Nothing abnormal by the low power; nuclei stain well; islets normal, no engorgement of vessels; with higher power the glandular epithelium is seen not to stain well and shows minute vacuoles. Many of the glandular epithelial cells by the osmic acid method show similar granules and droplets to those already described. The acini affected are not confined to any particular spot, but irregularly distributed, one group perhaps showing the change extensively while adjacent groups are very little affected, or almost normal. In some places it would appear that those cells nearest the vessels show the greatest change, but this is by no means general.

In this organ much of the deposit is rod-shaped and granular rather than in droplets.

Gastric mucous membrane: In one patient, G. P., there is a small area of denudation of the epithelium, but nothing else abnormal is noticed. In the other, J. P., no abnormality is detected.

Small intestine: In G. P. the mucous membrane appears to be generally normal; the nuclei stain well and the cells are distinct. In two situations in the section there is an accumulation of round cells beneath the muscularis mucosae, but no distension or engorgement of the vessels, no pus formation, no giant cells, no caseation,

no pigmentation. In another spot this heaping up of cells had thrust through the submucous coat to the mucous layer, which is being denuded.

In J. P. there is considerable congestion of vessels beneath the mucous membrane and dilatation to form a picture of blood sinuses or extravasations into inflammatory connective tissue. In this patient there are several places in which the mucous membrane epithelium has been shed and small erosions found.

Large intestine: Shows well the localised accumulation of small cells with occasional shedding of the overlying mucous membrane. The submucous blood spaces are lined by endothelium, and are more distinct than in the small intestine.

Mesenteric glands: In G. P. the capsule in some of these is a little thickened, the gland tissue itself is hyperaemic and the vessels distended and engorged. In the case of J. P. there is similar congestion, but no thickening of the capsule is seen.

It may be stated here that sections of all these tissues and also of all the various parts of the nervous system were stained for organisms by various methods—Giemsa, Löffler, Eosin-Gram-Weigert, Gram, Carbol-fuchsin—but no organisms were detected.

We pass on now to the description of the various parts of the nervous system which were submitted to microscopical examination.

Median nerve: Marchi. G. P. Brownish-black and black deposits granular and in droplets; in some of the fibres mostly granular and in masses. A large proportion of the fibres are unaffected; here and there a fibre shows the black of the Marchi staining, but the fibres so affected are relatively not numerous. It is noticeable that there may be several in one bundle, while those in the vicinity show none. Even in those with the most staining, the deposit is more of the diffuse brownish stain and not as droplets of broken-up myelin. In transverse section it is quite rare to see a nerve fibre with a distinct black ring of degenerated myelin.

J. P. In this patient the nerve fibres themselves are even less affected. In transverse section it is noticed that the nerve fibres themselves are many of them untouched. Minute droplets are seen in the fibrous tissue and in the walls of the vessels.

Sciatic nerve: Bundles cut longitudinally show considerable

general brown staining; also black, granular and patchy deposit, at times in droplets. These, however, seem in many cases to be more of the nature of a deposition of the stain *on* rather than *in* the nerve fibres, and in bundles cut transversely it is comparatively rare to see a fibre showing the stain, while granules are visible in the perineurium and in the vessel sheaths. No congestion of vessels, nor increase in fibrous tissue is noticed.

Some of the fibres cut longitudinally do show fragmentation of myelin and droplet formation, but such are not very numerous.

Other methods of staining do not reveal any abnormality.

Ulnar nerve: G. P. The change here is much more extensive than in other nerves. The majority of the fibres in longitudinal section show fat droplets, some exhibiting large droplets in series along the whole length of the fibre shown in the section; others have smaller droplets scattered at intervals along the course of the fibres.

J. P. In the corresponding section in this case there is very little involvement, hardly any of the fibres show the large droplets and fragmentation, and only a comparatively small proportion reveal any degeneration at all; the majority of the fibres are unaffected.

Posterior Root Ganglion: Cervical: G. P. A majority of the nerve fibres in this section show a black deposit in granules and droplets, and fully half are extensively affected. Many of those cut longitudinally appear as a series of droplets along the course of the fibre, and of those cut transversely the axis cylinders are surrounded by a black ring of myelin.

The large ganglion cells are much affected; many of them reveal a protoplasm almost entirely converted into black dots, in the midst of which, by careful focussing, the nucleus can be seen; others show a more peripheral distribution of granules, the nucleus being quite distinct; while yet again others contain merely a few fine granules in some part of the cytoplasm, usually distant from the nucleus, which is eccentrically situated.

J. P. In this patient the corresponding sections, though showing analogous conditions, nevertheless exhibit them in much less degree. The proportion of fibres affected is comparatively small, and it is only rarely that one is seen containing large droplets, and of those cut transversely only 1 or 2 per cent. show the distinct black ring

round the axis cylinder. Also the ganglion cells show none of the grosser changes mentioned above; the utmost seen is a cell with an eccentric nucleus and a small patch of fine granules somewhere in the protoplasm. Many show no abnormality at all.

Dorsal: G. P. More deeply involved than the cervical root ganglion; very few fibres have escaped.

J. P. In this the condition is practically the same as in the cervical region as regards the fibres; the numbers involved are certainly not more than in the cervical (thus differing from G. P.) and the ganglion cells show but slight changes.

Spinal Cord. Cauda equina: The fibres of the equina show fatty changes to varying extent in different parts. In those showing the greatest involvement about 25 per cent. of the fibres are affected, but in most parts the proportion is much below this, only a small proportion of fibres being degenerated.

Lumbo-sacral cord: The posterior columns show considerable degenerative staining fairly generally distributed, but possibly a little more marked in the external parts, and only slight in the ventral field of the posterior root zone. The radicular zone is considerably affected also, but more towards the periphery. Lissauer's marginal zone shows much blackening also.

The antero-lateral ascending tract shows some involvement, and in the lateral ground bundle there is a slight, generally distributed black stippling and degeneration of the long and short association fibres. The site of the tractus rubro-spinalis shows practically no degeneration, and the anterior and lateral vestibulo-spinal tracts also are but slightly affected.

Dotting of degenerated fibres is also seen in the regions of the spino-thalamic and spino-tectalis tracts, and possibly in the corresponding descending fibres (thalamic and tecto-spinalis); the anterior tecto-spinal tract shows considerable involvement.

Little change is visible in the lateral pyramidal tracts or in the direct (anterior).

No affection, or very slight, is seen of the fibres in the anterior commissure, though numerous scattered droplets are noticeable in the fibres of the anterior ground bundle. The various groups of the cells of the anterior cornua are all involved, but the dorso-lateral, intermediate and ventral all to a greater degree than the medial.

Lower Lumbar Cord: The posterior columns consist largely of fibrous tissue with masses of fat. Some fibres have escaped, but unaltered fibres are by no means numerous. There is a little perivascular infiltration, and phagocytes are visible bearing fat.

There are fatty fibres dotted here and there, but less marked than in sections higher up. It cannot be said that any particular tracts are markedly singled out. The various tracts affected are the same as those described in the section of the lumbo-sacral cord, with one or two minor differences. Thus, the marginal root zone shows more staining, and possibly also the vestibulo-tecto-spinalis and thalamic tract areas. The cells of the anterior horn are fairly involved, but, as before, the medial group less than the others. Most of the cells contain collections of coarse granules and fat, though showing no obvious changes in the nuclei. The anterior commissural fibres show some staining.

Middle of Lumbar Enlargement: There is no appreciable difference noted between the affections of the tracts in this section and the last, except that the degree of involvement of the posterior columns appears more marked.

Upper Lumbar Cord: The posterior columns here show considerable fatty degeneration and fibrosis. The perivascular infiltration, which is quite noticeable higher up the cord, here is but slight, but leucocytes containing fat may be seen more or less throughout this part (posterior column). The remainder of the section shows scattered degenerated fibres. A similar condition is now present in the dorsal cerebellar tract, but less than in the antero-lateral ascending. The cells of Clarke's column mostly exhibit considerable granular degeneration and chromatolysis.

Lower Dorsal Cord: The relative fibrosis and the fatty degeneration of the posterior columns continue to increase as the cord is ascended; the perivascular infiltration again is less marked than in the upper and mid-dorsal regions. The beginnings of the direct cerebellar tract show fatty changes, and the cells of Clarke's column are very heavily charged with fat. There is some, but not very marked involvement of the ventral part of the posterior root zone, while those fibres of the posterior columns entering the root zone medial to the posterior horn (? Schultze's bundle) are more affected. The postero-medial root zone and the marginal zone are markedly

involved. The crossed pyramidal tract shows very little change, as does also the direct. The fibres contiguous to the sulcus, the fasciculus sulco-marginalis (tecto-spinalis anterior), show definite fatty degeneration.

The areas of the anterior ground bundles show scattered droplets. The cells of the anterior horns are definitely stippled, and those of the dorso-lateral and intermediate groups rather less than those of the ventral and medial.

Mid-dorsal Cord: The posterior columns are still more extensively affected; there is much apparent fibrosis, partly caused no doubt by the disappearance of some of the fat, but the tissue entangles, as it were, large globules of fat. In some sections numbers of endothelial cells containing fat are seen, and there is perivascular infiltration with fat-bearing cells. The posterior root bundles are very fatty, but not so deeply affected as Goll's column itself. The direct cerebellar tract is severely involved, fully half the fibres appearing degenerated. The crossed pyramidal tract shows only occasional fibres with fatty changes. The antero-lateral ascending has the majority of its fibres attacked. The cells of the anterior horn show, many of them, fatty degeneration, and this change is present to quite the same extent, in fact more, in the cells of Clarke's column. The marginal zone shows a mass of black fibres. In the lateral ground bundles only scattered fibres are seen affected, but the anterior marginal bundle (? vestibulo-spinalis) is badly degenerated.

Upper Dorsal Cord: The fatty degeneration is so marked that it is difficult to make out whether one part is much more affected than another, with the exception of the following where the involvement is comparatively slight, namely, the crossed pyramidal tract, the lateral ground bundle and the commissural bundle. Of the remaining parts the most intense changes are seen in the posterior columns, the marginal and posterior root zones, the dorsal and ventral cerebellar tracts, and the anterior marginal bundle. Where the nerves appear also in the section it is noticeable that the majority of the posterior fibres entering the cord are markedly degenerated, as compared with the considerably smaller proportion of the anterior passing out.

Cervical Cord: The whole of the posterior columns show extensive degeneration, less marked in the ventral field of the posterior root

zone. The marginal root zone is greatly affected also. Mingled with the fat in the posterior columns is seen an excess of fibrous tissue, though some of this may be apparent only from removal of a portion of the fat. There is considerable perivascular infiltration, the perivascular lymph spaces being in parts almost choked with fat-containing cells.

The direct cerebellar tract, though extensively involved, shows a little less change than that in the antero-lateral. The lateral ground bundle is less altered than other parts, and here the area most affected appears to be the spino-thalamic fibres, and in the anterior ground bundle the corresponding fasciculus sulco-marginalis. The anterior vestibulo-spinal tract area is also deeply stained. The pyramidal tracts have not by any means escaped, but compared with the sensory areas there are only a few scattered fibres degenerated.

Cornual cells show much stippling with fat granules, more marked in the dorso-lateral, intermediate and ventral groups, less in the medial.

Medulla: There is marked degenerative staining of the funiculus gracilis, less, but still very distinct, in the funiculus cuneatus; the substantia gelatinosa shows very little change. The direct cerebellar tract is markedly affected; the crossed pyramidal tract but slightly. The cells in the anterior cornua of grey matter are only involved here and there, and then only minute dots are seen.

In the fissure of the upper part of the cord and round the central canal, sections of tissue treated by the Levaditi silver impregnation method, black, bacillary, rod-shaped bodies are seen. These, however, are probably only fat, as none of the ordinary methods used in searching for bacilli (Giemsa, Löffler, Eosin-Gram-Weigert, Gram, Carbol-fuchsin, etc.) showed any of these bodies, the preliminary treatment with alcohol and xylol having, as I interpret it, led to their solution and removal.

Pons Varolii: Except in the pale areas there is a general distribution of the fatty metamorphosis, but in small masses in the neuroglia, possibly due to the removal of the fat there, leaving the connective tissue more prominent. The cells themselves are fairly generally affected also, but in a minor degree, as a faint dusting of granules. The cells of the vestibular nerve root show considerable degeneration, mainly peripheral to the nucleus, both the descending root and the

principal nucleus. There is a patchy distribution of the staining in the general ground work, especially in the formatio reticularis The substantia gelatinosa shows practically none; while the fillet is considerably involved. There is slight 'dusting' of the cells of the VIIth and also of the superior olive, none of the central tegmental tract.

In the case of J. P. the deposit is very much less than stated above in the case of G. P. The groundwork shows some, but hardly any cells are affected at all.

Throughout the cord sections the description given is made mainly from examination of the tissues taken from G. P.; in the case of J. P. the distribution is seen to be similar, but the degree of involvement throughout is much less.

Cerebellum: In G. P. the cortical layer shows very occasionally cells with a few minute black droplets; the Purkinje cells are mostly intact, a few contain very minute granules and exhibit chromatolysis. In the interior—the nuclear layer—of each branch of the 'arbor' there is marked degeneration. Some of the vessels also contain fat-droplets. In J. P. a similar condition is seen, but in much less degree. Only one or two cells in the cortex are involved; no Purkinje cells appear to be affected; a few granules and larger fatty masses are seen in the inner (nuclear) layer, where the change is most marked in G. P. The vessels here, too, occasionally contain a few droplets.

Superior Peduncle: G. P. Patchy staining similar to that already described, affecting the groundwork, but the cells are nearly all involved, showing numerous granules and droplets; the nucleus, though indistinct in some cells, is still recognisable in almost every instance.

In J. P. the degree of involvement is quite slight, and is mainly confined to the white substance; a small proportion of the cells also is affected, but very small as compared with G. P.

Cortex Cerebri: Frontal. A considerable number of the large cells, varying in the different sections, exhibit the granular deposit, generally of the periphery, occasionally throughout the cytoplasm. In the latter case the granules are coarser and the droplets larger at the periphery. The fibres in the cortical grey matter are not affected, but in the white subcortical region there is extensive granular

patchy staining with the Marchi fluid. In the other case a similar condition of things is present, but less extensively distributed; there are fewer cells involved and less affection of the subcortical layers.

Motor Cortex: In this part the cells are much less affected both in degree and in proportionate number, but the deeper fibrous layers are more extensively involved than in the frontal region.

In both parts, whether the cells show merely a fine stippling or masses of minute droplets of fat, the nuclei of the cells appear to be unaffected, and further, although the white matter contains the droplets, as stated, all over, it is difficult to say whether it has any definite relation to the nerve fibres.

Optic Thalamus: A generally distributed black staining of the fibres is visible, but by far the most marked change is seen in the cells of the granular matter, which nearly all contain many large droplets, sufficient in many instances to obscure the nuclei.

Lenticular Nucleus: Appears to be practically uninvolved. Here and there one may come across a cell showing a few granules and a little chromatolysis, but such are far from common. The fibres in the neighbourhood show a patchy and granular deposit.

Corpora Quadrigemina: Many of the large nerve cells contain fat in somewhat coarse granules, which may more or less fill the cytoplasm. Throughout the section may also be seen a number of fatty nerve-fibres. Even those cells which contain many granules seem for the most part to retain their nuclei intact.

Hypophysis: Sections of this organ show extensive granular staining with the osmic acid. Every cell apparently has granules, either few or many. The fibrous elements, however, and also the vessels and capillaries exhibit none.

Auditory Nerve: Several fibres contain very small granules, in fact, the majority, while the grey matter shows fatty changes in the large nerve-cells, but not marked.

Optic Nerve: Widespread degeneration distributed along practically all the fibres; very few have escaped.

In the sections of the optic chiasma the large nerve-cells contain in some cases masses of fine fat granules.

Olfactory Lobe: Sections of this reveal considerable affection of the nerve fibres; the large cells, however, are very slightly involved;

here and there may be seen a cell with black granules and droplets, especially at the periphery remote from the nucleus.

Microphotographs are appended to illustrate some of the points mentioned above.

VII. DISCUSSION

Having given accounts of the histories, symptoms, physical signs, and morbid anatomy of this condition, we are justified now in discussing the important question: 'Are we dealing with a new disease?'

The question will be dealt with under the following heads:—

1. Is it Pellagra?
2. Is it Beri-beri?
3. What relation, if any, does it bear to the so-called 'Peripheral Neuritis' in Jamaica?
4. Is it a new 'deficiency' disease?
5. Is it microbial in origin?
6. Is it an intoxication?

These questions are somewhat interwoven, but in order that the matter may be presented as lucidly as is in my power, and afford a basis for future discussion and investigation, they will be kept separate as far as possible.

1. In the ensuing discussion as to the probability, or even possibility, of the condition being pellagrous, I have made free use for comparison of the report of the Second Triennial Meeting of the National Association for the Study of Pellagra (1913), and I beg to preface the points about to be brought forward by acknowledging my indebtedness to that report.

Firstly, as regards the onset. In Pellagra, the report states (p. 41): 'on account of the vague nature of the early symptoms of the disease it was impossible to determine the exact date of the onset of the first attack in all of them. The year of onset, however, was determined in 317 of the 323 cases, but the month of onset was determined with a fair degree of accuracy only in 181."

Now, in contradistinction to this, in the Spanish Town cases, not only was the year or month certain, but the actual day and hour of onset known. Also, in none of them could any history be obtained of a previous attack in the same patient; there is only one which

either terminates fatally, or becomes chronic, or clears up entirely, or, lastly, partially clears, leaving residual symptoms.

In pellagrous districts, as recorded in the report, the largest number of cases in which the month could be determined occurred in May and June, with a gradual rise to the maximum, then a gradual decline during the succeeding months. Whereas the majority of the Spanish Town cases occurred in March, April and early May, and then the presentation of fresh cases suddenly ceased (a likely explanation of this will be given later).

As predisposing causes of pellagra, it is mentioned that the patients lived in squalid surroundings, were poor, overworked and underfed (p. 73). Although some of my cases at Spanish Town lived in squalid surroundings, they were certainly not overworked nor underfed. Almost without exception they said that they had had plenty of food, usually yam, bread-fruit, and such like, but the cane crop being then cut and carried, they were living practically exclusively on cane. As regards food, the pellagra report states: 'Despite the rural location of the homes of the pellagrins of the agricultural class, they, too, were found to depend to a considerable extent for their food upon the country grocery store. It was the exception to find a pellagrin who could state that even the major part of the food used in the family had been home-grown.'

'Much of the food had undergone some process for its preservation, such as desiccation, canning, etc.'

In my cases the reverse conditions prevailed. In ordinary times they relied (with the exception of salt-fish obtained at a store) upon home-grown articles, yam, bread-fruit, cocoa, peas, beans, and so forth, and, as already stated, during the crop-time they lived on sugar-cane. Grimmon gives the following in his summary (p. 49):—

Race: More cases developed among the whites than among the negroes.

In the Spanish Town cases no white man was attacked at all; only West Indian labourers on the cane fields.

Sex: More cases occurred among the females of both races than among the males.

The distribution was practically equal at Spanish Town.

Age: More cases developed at ages between 20 and 40 than at other ages.

This also obtains in my cases; in fact, all of whom I have detailed

records were adults except one, a girl of 14 years, and she was earning her living by labouring on the estate.

Date of Onset: More cases had their onset during the months of May and June than in other months.

This has already been referred to above.

Relationship of Cases: More cases developed in the vicinity of other cases than otherwise.

This point is not of much use as positive evidence, it is equally in favour of a common source of origin.

Heredity: None of the facts seem to indicate that pellagra is hereditary.

On p. 113, Professor Victor Babes states: 'In spite of the rich literature on pellagra, scarcely so important a malady exists about which we are so badly oriented. One thing only seems to be established about its etiology: that it belongs to the disease of poverty or insufficient alimentation, and notably that it is in intimate relation with nutrition by maize of bad quality.'

With regard to the Spanish Town cases, they occurred, it is true, amongst the poor, in so far as labourers are not well-to-do, but they were not destitute; although the cost of food was high at the time, this fact did not affect them to any appreciable extent as they lived on the cane which they took while at work, and if they had anything else it was merely the usual ground provisions; they do not eat maize to any extent, particularly in the cane season; at other times they use it as cornmeal. Practically all said that they were getting plenty of food.

Of fifty cases of pellagra in which the history of the onset was obtainable, only one of those recorded in the pellagra report (p. 121) bears any resemblance to the Spanish Town cases, and that is not a close one. The account states that the disease began 'just before Easter, weakness, disinclination for work, tingling sensation in the hands and feet. At the end of a week, erythema.'

In my cases the onset was in all instances sudden, there was no previous weakness or disinclination for work, that is to say, no abnormal disinclination (of course, it must be remembered that all West Indian natives, as soon as they are old enough to work at all, exhibit a disinclination for work). Eye or mouth symptoms, or both, without exception, preceded any nerve symptom; these latter were

not tingling, but numbness, and began in the feet several days, perhaps two to three weeks, before any similar symptoms appeared in the hands; and, lastly, in none of them did any erythema follow, even when watched for months.

Again, one of thirty cases of pellagra described in detail by Dr. Deeks (p. 182) bears a superficial resemblance to the Spanish Town cases:—'L. B., Martiniquan, coloured, male, aged 40, admitted July 13th, 1910, complained of having been sick for eight days and unable to work because of pain in the joints and extremities. On admission the temperature was 110·4° [probably a mistake for 101·4°], pulse 128, and respirations 54. Patellar reflexes absent; Romberg's sign present; gait ataxic; unable to coordinate. He had the characteristic oral mucous membrane but no dermatitis. From the mid-day of his admission he vomited almost constantly, was nervous, slept badly, and developed an uncontrollable diarrhoea; temperature increased, and he died with a temperature of 106° on the fourteenth day after his admission.

Leucocytes on admission: polymorphonuclears 81 per cent., large mononuclears 4 per cent., small mononuclears 15 per cent.'

The points of resemblance are: (1) The patient was ill for eight days before any nervous symptoms presented themselves. (2) Absence of patellar reflexes. (3) Ataxic gait and incoordination. (4) No dermatitis, but sore mouth. (5) Diarrhoea towards the termination.

The points of difference: (1) Pains in the joints and extremities; the former was slight in some cases of the Spanish Town series, but not definite pain even then, and movement was always quite free and painless. (2) Rise of temperature, rapid pulse and respiration; all of these are absent in my cases. (3) Romberg's sign is absent, when the patient can stand well enough for this to be tested. (4) Vomiting was never a symptom in any case.

In short, except for a few symptoms, there is nothing else common to both, and the general picture is quite different.

Of the blood changes in pellagra, Hillman writes: 'Examination of the stained preparation of the blood revealed very little change in structure or staining reactions of the red cells; some irregularity in size was noted in a few cases, microcytes appearing to be the most common form. No nucleated cells were seen.

'The differential leucocyte counts showed the most important and constant alterations (500 cells were counted as routine). . . The cells increased were chiefly the small lymphocytes. Including both large and small types of lymphocytes, the percentages in the more pronounced cases ran as follows:—43·00 per cent., 36·00 per cent., 50·35 per cent., 47·20 per cent., 52·00 per cent., 41·80 per cent., 40·00 per cent.

'The majority of the pellagrins show a relative increase in the lymphocytes at the expense of the polynuclear neutrophiles. As to the large mononuclear leucocytes the differential counts show, on the whole, a slight increase in most cases, rising as high as 5·6 per cent. in one instance.

'The polynuclear eosinophiles maintain a normal average in most cases, but in three were in excess, amounting to 8·00 per cent., 7·80 per cent., and 5·40 per cent. Other causes of eosinophilia were not demonstrated in these patients. The average eosinophile count in this series (twelve cases) was 2·86 per cent.'

Summing up, he states 'The increase in lymphocytes is interesting, and what one might expect in a disease associated with a disturbance in gastro-intestinal function and structure . . . In pellagra we probably have to deal with a disease associated with a rather low grade toxin of some nature, which, in common with many other pathological states, is absorbed by the lymphatic system and through local irritation of certain lymph nodes produces an increased circulatory activity in these situations, in consequence of which large numbers of lymph elements are swept from the lymphatics and enter the general circulation. This is in accordance with Ehrlich's theory of lymphocytosis. The large mononuclear leucocytes are not sufficiently increased to warrant drawing any definite conclusions.'

The blood findings and blood picture of the Spanish Town cases have already been dealt with in some detail, and need not be repeated again here. A comparison between Hillman's findings and mine may most easily be made by a perusal of the Table I of this report, when the resemblances will be seen to be considerable.

The nervous histo-pathology of pellagra has been ably described by Singer and Pollock, and a comparison of their findings with those given above of tissues taken from the cases of the Spanish Town disease will show that there are many points of resemblance.

Pia mater: In their cases there was some degree of thickening with an increase in fibroblasts and an infiltration with cells which were derived mainly from the vessels, belonging to both the endothelial and the adventitial types. In some there were a few lymphocytes, and occasionally extravasated erythrocytes. They found as a constant feature the presence of pigment granules. The change was most marked over the convexity of the brain in the central, frontal, and temporal regions, and particularly in and adjacent to the sulci. In the latter situation especially they describe a growth of glial fibres into the pia.

In the larger arteries there was hypertrophy of the muscularis and thickening of the vessel walls, while the smaller vessels showed proliferation of the cells, endothelial, muscular, and adventitial, and some degree of perivascular infiltration made up of lymphocytes and vessel cells.

In their cases the difficulty naturally arose of distinguishing to what extent the changes were due to the pellagrous condition or to the accompanying mental or nervous disease. The vessels showed a moderate proliferation of the cells of the adventitia and media, and, in a few, there was an increase of the intima. The muscle cells were swollen, distorted, and contained granules of pigment. In the sheath they noted fatty and fibrinoid pigment. The perivascular infiltration was not marked.

There were similar changes in the vessels of the spinal cord, and a more or less definite increase of connective tissue along the septa. Dilatation of the small vessels in the grey matter of the cord was marked in some of the cases. This is very distinct in some of the Spanish Town sections. The neuroglia fibres were increased at the outermost layer of the cerebral cortex, and in the spinal cord about the central canal. They state that the satellite and the Trabant cells were increased about the ganglial cells, which showed the true Nissl changes.

The glia cells of the outermost layer of the brain cortex were frequently seen distorted, darkly staining and shrunken, showing intranuclear metachromatic granules. The small cortical glial cells were increased in number, and at times were noticed to form considerable masses about the vessels. In all cases some degree of fatty degeneration of the glial cells was found. The central canal

was occluded in all cases; this is not abnormal, and is seen in some of the sections of the Spanish Town cases. In their cases, however, the occlusion was not due merely to general débris and degenerated epithelial cells, but to a definite proliferation of the glia and ependymal cells, constituting to all intents and purposes a central gliosis.

This was not seen in my cases.

The authors affirm that the axonal reactions in the nerve cells are more definitely related to acute pellagra. The cell is swollen and rounded, the nucleus displaced to the periphery and may even be extruded. The tigroid substance has mostly disappeared from the central portion of the cells, blocks staining well being often left around the periphery and especially in the base of the larger dendrites, a considerable mass being often found around the nucleus. The nucleus is distorted, often oval or reniform, and stains more or less uniformly, with a pale colour.

Axonal changes are less marked, and may be quite absent in long-standing cases or in the interval cases. It is less noticeable in a patient dying three and a half months after an attack, and absent in one dying eighteen months after.

These were not conspicuous, however, in the Spanish Town cases, in those dying after a brief illness.

They further state that in recent cases axonal changes were severe in all, and in many involved practically all the Betz cells and many of the large pyramidal cells of the central convolutions. They found similar changes in the ganglion cells of the hippocampus, the dentate nucleus, the central ganglia, and the nuclei of the cranial nerves.

In the cord these changes in the cells of Clarke's column were often extreme, and in the anterior cornual cells in two-thirds of the cases, in the posterior cornua in one-third, and in one of them more marked than in the anterior. Similar reactions were found in the posterior root ganglia, and in the cells of the semilunar ganglia of the sympathetic and of Auerbach's plexus in the intestine.

Some Purkinje cells showed central chromatolysis, but none the true axonal change.

'The cell showing axonal reaction is, except in very severe degrees, capable of recovery, and the above facts would suggest that

the change is the result of the condition, whatever it is, which underlies the acute pellagrous outbreak. During the intervals the cells again resume, to a greater or less extent, the normal state. This type of reaction also seems to result from an injury to the neurone at a distance from the cell, and such an hypothesis is well borne out by the absence of satellitosis about these cells.'

They describe six types of Nissl changes, all of which are most marked in the central and paracentral convolutions, but are also present in various regions of the cortex. These types are :—

1. The cell-body may be shrunken and distorted, staining more or less uniformly pale, with a nucleus rich in chromatin.

2. Similar bodies with pale nucleus of the same or lighter colour than the cytoplasm, and containing bodies stained bluish-green with thionin.

3. Shrunken uniformly dark-staining cell-body and a dark nucleus which may almost fill the cell.

4. Simple chromatolysis, the body being of normal size or a little larger. The Nissl granules may be pale and rarefied, or present merely as dust. The nucleus has a dark nucleolus, but is itself approximately normal in staining and position.

5. As a more extreme degree of the last, where the outline of the cells is indistinct, the nucleus is absent—a mere shadow cell.

6. Vacuolated cells with a nucleus, but showing no Nissl bodies.

Pigmentary changes were present in all their cases and in all the various types of cell-changes, even in those showing normal staining, but marked in the Betz cells, the large pyramidal and other cells showing axonal changes. The pigment stains black with osmic acid, and in some cells within the nucleus there may be basophile and fibrinoid granules.

It must not be forgotten that some degree of pigmentary deposit of a fatty nature is to be considered as a normal condition—a result of failure to remove all metabolic products, whether due to faulty elimination or excessive production, or they may be the result, as Victor Babes has suggested, of degeneration of the chromatin material of the cell.

In pellagra, however, they far exceeded the amount present even in old age, and so far they must be regarded as pathological.

Nerve fibril changes consisted of agglutination, fragmentation, and loss of fibrils, which is exceptional in pellagra. The fibrils are now regarded as of great importance in the performance of the nervous functions of the neuron. Complete loss of fibrils, would, therefore, entail loss of function, and probably a consequent impossibility of recovery of the neuron. In all their cases a marked paucity of processes was noticed in the larger cells. The architecture of the cortex was not much disturbed, in the deeper cell layers of the Rolandic region there was some distortion of arrangement and a paucity of cells. This was noticed in the Spanish Town cases.

Marchi-treated tissues showed in the brain a diffuse, scattered degeneration of the radial cortical fibres, but not of the supraradiary or tangential. In the spinal cord there were 'degenerated fibres in all regions, not in any way systematic, involving perhaps to the greatest extent the posterior columns, and especially the subpial fibres in two cases. The crossed pyramidal tracts were not more affected than other regions, and in some cases were practically free.'

The anterior and posterior roots showed a few degenerated fibres, sometimes more marked in the cervical, sometimes in the lumbar region.

In sections of the cord the loss of stain in Goll's column was most marked in the centre, bordering the middle line. In a few it extended forward to the commissure, and in some back to the posterior surface where it outlines exactly the whole column. Under a high power the degeneration was seen to affect quite scattered fibres, the majority being well stained; and from its situation seemed to involve the endogenous fibres rather than the main ascending tracts. It was most marked in the cervical region, and might in some cases be entirely absent in the lumbar and lower dorsal. In two cases a similar pale staining was found in the crossed pyramidal tracts, in three in Lissauer's tract, in one in the lateral basis bundle, and in two in the posterior ground bundles.

Older authors claimed to find definite systemic degeneration, generally a combined postero-lateral sclerosis. Later work, however, seems to show clearly that systemic degenerations are certainly exceptional and were not present in the cases studied by Singer and Pollock.

Amyloid bodies and pigment granules were found in excess in all cases, and might be numerous both in brain and spinal cord. They were described beneath the ependyma, along the vessels, in the thickened glia of the most superficial layers of the cortex, and the periphery of the spinal cord, and also around the central canal. Though their significance is not clear, they undoubtedly occur, especially in chronic degenerative conditions.

Their origin is not yet determined, so far as I am aware, but they are considered to represent degenerated axis-cylinders or myelin sheaths (Siegert Wolf) or glia cells (Obensteiner) or to be degeneration products occurring within the glia-cell body, which are at first surrounded by a layer of fat, and on the disintegration of the cell they are set free and the fatty circumference is dissolved; again, they may result from the precipitation of certain materials from the tissue juices by the fixing fluids, and thus be in some degree a post-mortem artefact.

If the above description of the pellagral nervous tissue findings be compared with what has been written earlier of the findings in my cases at Spanish Town, it will be seen that there are indeed remarkable points of resemblance, especially in the generalised distribution of the degenerative changes in the spinal cord; but, as stated in my description, though not exactly picking out any definite system in the cord, the changes throughout are much more marked in the sensory conducting tracts than in the motor, and the symptoms present would lead one to expect this.

It must be remembered that free pigment granules of a fatty nature in the tissues and perivascular lymph spaces, and the pigmentary degeneration of nerve-cells, are not signs of any one definite disease, but are merely indicative of some more or less chronic degenerative condition. Before summing up the matter as to whether the Spanish Town cases may or may not be acute pellagra, I may quote the general résumé of Singer and Pollock on their own findings in the latter condition:

'The one feature which seems to bear the most definite relation to the acute pellagra attack is the axonal change of particular nerve-cells already discussed. With this is probably to be related the scattered Marchi degeneration found throughout the nervous system. This particular picture of widespread axonal chromatolysis with

special involvement of the Betz cells and those of Clarke's column has been noted by all workers upon pellagra with modern methods of staining (Marinesco, Babes, Lugardo, Anderson and Spiller, Kozowski, and others). As has already been noted by one of us, this picture is identical with that described by Meyer as central neuritis. In a personal communication this author assures us that none of his cases presented clinically a picture of pellagra. Attention may also be drawn, in this connection, to the publications upon this syndrome, and upon the cytology of the brain in mental disorders by Southard, Corist, Cotton, Somers, Orr and Coles. In a case of chronic alcoholism recently studied by us, there was, clinically and pathologically, a picture of central neuritis and yet nothing to suggest a history of pellagra. As a matter of fact it would be impossible to distinguish microscopically between the specimens of this case and those here described. We must therefore conclude that pellagra is not capable of recognition post-mortem, apart from the presence of the typical lesions. This is contrary to the opinion expressed by Kozowski in his very detailed report of cases with an excellent digest of the literature.

'The interpretation of these facts is possible only in one way, namely, that such a picture as that presented in central neuritis is a mode of reaction to some harmful agent circulating in the blood, acting upon the axis-cylinder process of the neurons somewhere in their course. Further, it seems clear that this change may be brought about by various ultimate causes, although it is conceivable that the actual excitant of the reaction is the same in all and a product of body metabolism under morbid conditions.

'Besides these acute changes we have also demonstrated the presence of more chronic nerve-cells and glia changes (chronic Nissl change, pigmentary changes, satellitosis, and the presence of an increased number of astrocytes, many of which show various forms of change), which indicate the existence of more chronic type of reaction. That this is an active state at time of death is shown by the presence of ameboid glia cells.

'These changes also differ from the axonal reaction in that they suggest a direct, or primary, action upon the nerve-cells. It is very difficult to determine the exact relationship of these changes to pellagra, but since they are apparently constant, even in the interval

cases (these, however, are not uncomplicated), it seems possible that there is a more chronic degenerative and toxic basis, interrupted at intervals by more acute exacerbations. A study of uncomplicated cases of pellagra dying during a free interval would be necessary in order to throw much light upon this problem.

'We do, however, feel justified from the facts here recorded in drawing the conclusions that pellagra is accompanied during the exacerbations by a generalised intoxication, and that there is nothing specific in the histologic picture presented.

'Conclusions

'1. Pellagra is accompanied by a general intoxication during the acute exacerbations.

'2. In common with other intoxicative conditions pellagra gives rise to a "central neuritis" reaction.

'3. Some of the changes are characteristic of this particular form of intoxication.

'4. There is no evidence of a local infection of the nervous system.'

Finally, then, as regards the possibility of the Spanish Town cases being pellagrous: Pellagra sine pellagra can, one supposes, occur, though the diagnosis of such must be more of the nature of a lucky conjecture than a certainty. Taking it as a general working rule in pellagra that the sequence of events is usually gastro-intestinal symptoms first, cutaneous manifestations second, nervous and mental symptoms third, nevertheless there is no doubt that the order is not by any means invariable, for sometimes the skin manifestations may be the first recognized, while again, in a smaller number, the nervous and mental symptoms appear to precede the others. Close questioning, however, will often reveal the fact that symptoms, possibly quite mild, had occurred one or more years previously.

Against the view of this condition belonging to such a category of pellagra sine pellagra with nervous symptoms as the primary manifestation are the facts that none of the patients exhibited any mental aberration whatever, that in one only was any skin affection present at all, and that was constituted merely by a loss of

pigmentation at the lower end of one radius around an old scar. In none of the other cases, and they totalled fully a hundred, was any cutaneous manifestation found, pellagrous or otherwise, and it is, to say the least, extremely unlikely that there should be a sudden outbreak of pellagra sine pellagra affecting so large a number of persons.

2. *Is the disease Beri-beri?*

We may take for the purposes of this part of our discussion the definition of beri-beri as given by Vedder (1913):—

> 'Beri-beri is an acute or chronic disease, characterised by changes in the nervous system and particularly by a multiple peripheral neuritis, with an especial tendency to attack the nerves of the limbs, the pneumogastric and phrenics. Ordinarily the clinical picture of a peripheral neuritis is combined in varying degrees with cardiac disturbances, oedema, serous effusion, and gastro-intestinal derangements. Exceptionally cases occur in which cardiac dilatation and sudden death are the first symptoms observed. It is a disease resulting from faulty metabolism, usually only seen in those persons who eat rice as a staple article of diet, and is directly caused by the deficiency of certain vitamines in the food.'

Vedder also states (p. 303) that 'peripheral neuritis *per se* cannot be the essential lesion in beri-beri, because degeneration of the nerves occurs before symptoms arise, because advanced degeneration may be present accompanied by no symptoms at all, and because degeneration of the nerves remains long after recovery has occurred.'

To resume. Firstly, as regards the symptoms: Whether we consider that the degeneration and paralysis of the muscles affected are the direct result of the neuritis, or whether we take it that the peripheral neuritis *per se* cannot be the essential lesion in beri-beri, it is an established fact that there is a certain predilection for certain muscles to be affected in this disease, and not all the muscles of a limb.

In the Spanish Town cases, on the contrary, it has been definitely stated that there was never seen any wasting of groups of muscles,

and until late no muscular atrophy at all, but as the disease was tending towards a fatal issue a general emaciation came on.

In fact, the motor system was not affected, to any extent at least, in any of the Spanish Town cases, and there was no appreciable loss of power in the muscular movements, even comparatively late in the disease, in fact not until general weakness and exhaustion supervened. With respect to other muscular symptoms, in none of my cases was there seen either contraction of muscles, painful cramps, tonic convulsions or fibrillary twitching, any and all of which may occur in beri-beri; moreover, there was no indication of the reaction of degeneration.

The two conditions agree in that the deeper reflexes tend to disappear early. In beri-beri there may be an exaggeration of the knee-jerk during the first week or so, and this has its counterpart in the case of M. H. of my series; while the superficial reflexes are normal.

Again, in beri-beri ataxia is not usually present; there may be, it is true there is, instability, but it is due to muscular weakness and not to incoordination. The anaesthesia, to take another symptom, in beri-beri most commonly starts over the tibiae, thence passing over the inner surface of the legs and calves and on to the dorsum of the foot, the wrists perhaps being affected at the same time, whereas in the Spanish Town cases invariably the condition started in the toes and soles of the feet, and extended gradually upwards, and later, if the upper extremities became involved at all, it was always in the tips of the fingers that the numbness was first noticed.

Other sensory symptoms are usually present in beri-beri which were never found in the disease at Spanish Town; for example, tenderness of the muscles, especially of the calves on pressure, is complained of in almost all cases, so that patients object and shrink from the muscle-pressure test in beri-beri; this did not occur once in all my cases, nor did any of them exhibit the circumoral anaesthetic area which is found in a certain number at least of beri-beri cases.

Passing on to the other symptoms; in beri-beri cardiac symptoms are nearly always present at some time or other in the course of the disease, such, for example, as palpitation, dyspnoea, increased action, pulsation in the neck, precordium, or upper part of the abdomen; an increase in the area of cardiac dulness, a tendency to oedema of

the lungs. Not one of these symptoms declared itself in the Spanish Town cases, and their absence can hardly be satisfactorily explained by saying that all cases (more than a hundred) were probably the 'dry form' of beri-beri.

Secondly, turning for a moment to the pathological conditions; in beri-beri, though the changes in the spinal cord may be fairly generally distributed, they may be but slight, in fact, the findings never seem to be so marked as those described in the section on the morbid histology of the tissues taken from the Spanish Town patients. Although we may agree that 'dry or paralytic beri-beri is caused by a defective metabolism resulting in more or less degeneration of the entire nervous system,' as Vedder states, nevertheless, personally, I have never seen the changes in the central nervous system in a case of beri-beri so much 'more' as to equal even the least of those present in the fatal Spanish Town cases.

A few words as to the blood conditions, for these do in a measure resemble one another. Though varied results have been reported by different authors, the present decision appears to be that in beri-beri there is not much change in the red cells, not more than might be expected in any serious disease, and that there is nothing characteristic in the differential leucocyte count, but that the Arneth count may be characterized by a slight shift to the right, Classes IV and V constituting some 34 per cent. of the whole.

In none of my cases was the latter seen, the highest being 21·3 per cent. and the majority about 15 per cent.

To sum up this part of our discussion, then, we may say that in spite of a few superficial resemblances, the weight of evidence against the beri-beri idea is overwhelming. In no case was there any oedema at all, nor any palpitation, or dyspnoea, in fact no heart symptoms or physical signs; in no case was there any tenderness of muscles, nor any true paralysis, no distinct foot-drop, no wrist-drop; the site of origin and direction of spread of the anaesthesia was different; in none was there any circumoral anaesthesia; the cord changes, as revealed in the central nervous tissues taken just after death, were most extensive and profound; and, lastly, rice, if it entered into the diet of these patients at all, did not constitute a regular or large part of it.

The next point to be taken up in our discussion is:

3. *What relation, if any, does the Spanish Town outbreak bear to the so-called Peripheral Neuritis of Jamaica?*

The first records that I can trace concerning this so-called peripheral neuritis, which is a very common condition in Jamaica, are articles by Dr. H. Strachan (1888 and 1897). The author of these was then Senior Medical Officer in Jamaica, and, as these articles show, a man of keen observation.

On reading his articles by the light of present-day knowledge, one sees that he has included cases with skin lesions and pigmentation which would now be regarded as pellagra, and other patients exhibiting local paralyses and muscular atrophies, which are not observed in those cases relegated at the present day to the category of this obscure peripheral neuritis.

The symptoms of a patient suffering from the so-called peripheral neuritis are as follows:—He has usually been ill for a long time, weeks, months, perhaps a year or two, and does not remember the nature of the onset in most cases. At the time when he presents himself at the hospital the chief complaints are numbness and cramps in the feet and hands, dimness of vision, and hardness of hearing. If the illness is of shorter duration, there is slight excoriation at the edges of the eyelids and margins of the lips, and the palpebral conjunctiva is hyperaemic. The degree of impairment of vision is very variable, it may be a mere dimness, or it may be such that the patient cannot count fingers, and may not be able to recognize his friends by sight. The deafness also may vary from merely a little 'hardness of hearing' to complete inability to hear at all.

'Sensation is blunted, but never completely abolished,' says Dr. Strachan. 'It is the numbness and tingling, or "crampiness" which the patient first notices . . . and which, if he be of a superior grade of intelligence, leads him to seek advice; if he be not, or be careless, it is the gradually increasing impairment of vision which brings him to the doctor.'

'There is,' he states in another place (p. 478), 'no alteration in the reaction of the pupil to light and accommodation, no falling when the eyes are closed, and the sphincters will not be affected.'

He mentions that in very grave cases 'the innervation of the diaphragm and of the heart is seriously involved . . . and leads to a fatal termination.' Here, I feel sure, he was meeting with beri-beri neuritis amongst the number.

He says again (p. 482), that 'redness and irritation of the eyelids and lips are often the first external signs noticed. It soon passes into a slight eczematous condition, especially at the corners of the mouth.'

Strachan thinks that the cause is malaria; he states 'as to the poison which, circulating in the blood of the affected person, causes this form of peripheral neuritis, I have been led to think that it is the poison of malaria.'

At the present day, particularly in certain parts of the country, many of these so-called peripheral neuritis cases may be met with, some going about, some in the hospitals, some in the Poorhouses. Those able to move about complain chiefly of 'dark sight,' which they interpret as inability to sew or read, or, in advanced cases such as one finds in institutions, inability to count fingers or recognize faces; they are nearly always partially deaf, in some cases quite deaf; those who can walk do so with a kind of 'steppage gait,' but without real drop-foot, and not with a tabetic stamp, nor on a wide base. They exhibit little, if any, loss of power until they become bedridden and emaciated, and at no time do they show any localized wasting of muscles or muscle-groups; the sphincters are never involved, the pupils react normally to light and accommodation; shutting the eyes does not increase the ataxia, the knee-jerks are absent.

Lieut.-Col. W. S. Harrison, R.A.M.C., when in Jamaica in 1914, made some brief notes on cases seen by him, and he states: 'The legs are always much more affected than the arms. Dimness of vision is a constant and early feature and may amount to almost blindness; deafness is also all but invariable, and the amount of it seems to vary with the severity of the disease. In some improvement may occur so that the patients are able to get about with a very ataxic, high-stepping gait, and then the condition comes to a standstill. Cases in the Almshouse have been there for six or more years with practically no alteration for four or more years.'

'Girdle pains are much more frequent in the early stages.'

He describes one chronic case which terminated fatally; the man

gave a history of the onset as 'fairly sudden with tingling and numbness in the feet, rapidly extending in the legs and to the arms, and he had been in this condition for at least six months.'

He noted particularly that 'there is comparatively little wasting of the muscles considering that the symptoms seem to point to a neuritis, but this is a constant feature of these cases. This man's legs were no more wasted than one would expect to see them in a man who had lain for six months in bed. There were no deformities from contractures, there were no skin lesions, the knee-jerks were absent, and the plantar reflex present, although the patient told me that he could not feel when I touched the sole of the foot.'

Harrison made the following general note after seeing a large number of cases which were got together for him as typical cases of peripheral neuritis. 'There were no children among the number. Paralysis varied a good deal in different cases, from a slight ataxia to complete inability to move the legs; in no case were the arms more severely affected than the legs, and, as a rule, it was only the legs which were seriously affected, although the patient complained of the numbness in the hands and that they often dropped things. The cases were of varying durations; some of the older ones having been disabled for ten years; deformities were conspicuously absent, while in no case was there any marked wasting of the affected limbs.'

After these more general statements he briefly quotes the following two cases: 'A young man, aged 27, who had been ill since Easter (about two months); the illness commenced with numbness of the feet, but he had had "soreness" of the eyes for some time before and gradually increasing dimness of vision. There had been no particular pain at any time, and no trouble in the arms. The eyes were now better, but the legs were getting worse and there were cramps in the feet. There was some deafness, but this was not very marked. Patient said he was now (May 29th) beginning to get "cramps and burning" in the hands. There was no loss of sensation, no atrophy of muscles, which appeared to be rather ataxic than actually paralysed. The wife of this man was also commencing to suffer from dimness of vision and burning of the soles of the feet, but no deafness so far, and walks all right, though soon tired. Knee-jerks absent.'

I have quoted this last note fully because it appears to me to

compare, even in many small details, with the cases of the Spanish Town epidemic, and during the last few months I have made a point of seeing cases of 'peripheral neuritis' when the opportunity arose, and the condition in practically all was that described above—numbness of feet and legs and difficulty in walking, slight deafness and 'dark sight,' numbness in some cases of fingers, with inability to sew and a tendency to drop things, and on examination absence of knee-jerks, retention of superficial reflexes, no objectively discoverable change in sensation, except in some a difficulty in distinguishing heat from cold; an ataxic gait, often steppage, but not tabetic, and without atrophy of muscles.

These patients may live for many years and die from some intercurrent disease, so I have not yet had an opportunity of examining the tissues post-mortem.

As a rule the patients have been so long ill that they have forgotten the actual symptoms at the time of onset; the numbness and ataxia remaining are most impressed upon them, and thus they come to regard these as the first symptoms. If, however, the memory is sufficiently good, or if the disease has not been so long continued, questioning as to eye and mouth symptoms will often bring out the fact that these did precede the actual nerve symptoms, as in the cases recorded above by Harrison. In my opinion there is strong evidence to show that the Spanish Town cases were examples of the acute form or acute stages of the condition which has for years been called in Jamaica 'Peripheral Neuritis,' and I feel sure that the elucidation of the cause of the former will also clear up the mystery of the latter.

4. *Is the condition a new 'Deficiency' disease?*

The suggestion has been made on more than one occasion to attribute 'peripheral neuritis' to some diet deficiency, more so, naturally, since beri-beri has been shown to be so caused.

Such a suggestion, unsupported by any evidence, merely serves to cloak our ignorance and to baffle investigation.

The chief argument brought forward in support of the theory is that analyses of the articles of diet used by the natives in Jamaica show a relative deficiency in protein and excess of carbohydrates, and this is backed up by analyses of the constituents of the dietaries supplied in institutions in which these cases are found. These arguments, however, will not bear examination. In the first place

these articles of diet are in use by the generality of the people (I refer to the poorer natives) from infancy upwards and at all seasons, nevertheless children and young adults are not affected, but the labouring class. Secondly, the initial symptoms seem to declare themselves at certain seasons, namely, at the time of cutting and carrying of the cane crop, and during this time little if any of the usually employed native foods are eaten at all. Several overseers have told me that the labourers come to work without any breakfast because they cut cane for themselves as soon as they get to the fields and eat it for breakfast, and they eat it all day long, and take practically nothing else while the crop is on. Thirdly, analyses of the dietaries in the public institutions where cases of peripheral neuritis are found have no weight, because these cases do not *arise* in such institutions, but outside, and they come into them only when they are no longer able to work and support themselves; moreover, those patients who are only mildly affected and get well, and, in fact, any who show improvement at all, do so on these very diets which are impugned.

The analogy, therefore, which some practitioners here bring forward that beri-beri is a deficiency disease, and occurs in epidemics (such as that in Bilibid Prison, Manila, in 1901-2), and that this peripheral neuritis is similar, is a false analogy altogether, for the epidemic here did not arise in any institution but among healthy labourers on sugar estates in the neighbourhood of Spanish Town, and these were attacked suddenly while at work.

Catto in his last report (1916), when mentioning 'peripheral neuritis,' gives analyses to show that 'with the exception of peas and beans all the native products are strikingly deficient in protein substances.' He mentions in connection with this the diets used at the Kingston and St. Andrew Union Poor House, and in support of the disease being a deficiency one quotes Koch and Voegtlin, saying that 'a diet deficient—even comparatively—in protein constituents, though rich in carbohydrates, causes in the more highly organised mammals a very marked disturbance in the metabolism of the elements composing the nervous system.' And, again, in his conclusions, 'this restricted diet is of a nature likely to produce detrimental effects in the human organism, especially on the nervous system.'

Apparently, then, we are asked to believe that because (1) a diet

deficient in protein constituents causes a disturbance in the metabolism of the elements composing the nervous system, and because (2) the native products, except peas and beans, are deficient in protein substances, and because (3) cases of peripheral neuritis show changes in the nervous system, therefore peripheral neuritis is due to the native diet.

I may mention also that in the same report Catto quotes a statement made to him by the medical officer in charge of the Poor House, the diet of which institution he has given, saying: 'Some are admitted with very marked nervous symptoms; quite unable to stand. After some months they are able to walk with the aid of crutches; gradually they discard these and use an ordinary walking-stick. Usually a considerable amount of shakiness of the lower limbs persists. In less advanced cases the patients complain of numbness or cramps in feet and hands, and a great heat about the body, especially in the back. Frequently the condition subsides after several months' treatment.' All this, be it noted, on the incriminated diet!

'In other cases the patients are admitted in a bedridden condition and never leave their beds, gradually sinking and dying from inanition.' 'Diarrhoea is the immediate cause of death in some cases.' Compare this last statement with the histories of the fatal Spanish Town cases.

As a minor point one might add that if these Spanish Town cases are to be ascribed, as the practitioners in charge of them do, to poverty and inadequate food, that is, to a food deficiency, why should there be the same history of onset in each, an acute origin with eye and mouth symptoms? Moreover, as stated in those histories, the patients were by no means all poor, and many stated that they were getting plenty to eat.

5. *Is the condition microbial in origin?*

To the discussion of this point I fear I have only negative evidence to put forward:

1. Careful examination and cultural experiments were made from the discharges, the abrasions, and the lesions generally of the eyes and mouth, where the first lesions appeared to be, but nothing beyond the ordinary air and mouth organisms were isolated. One

must not conclude, of course, that some organism may not have started the condition and then have disappeared.

2. Cultures of the blood were made at various periods of the illness, but all remained sterile, both aerobic and anaerobic.

3. Cultures of the cerebro-spinal fluid made on various media at various periods from the earliest to the latest, and even just after death, have all remained sterile.

4. Cultures of the urine and faeces have yielded only disappointing results; very rarely was any organism isolated which is not to be found in normal faeces.

5. In none of the tissues taken at the autopsies, and practically every tissue in the body was taken and stained in various ways to show the presence of organisms—both bacterial and protozoal—was any organism detected. Levaditi's method was carried out on all the nervous tissues as well as others, but no treponemata or such-like organisms were seen.

6. The results of the detailed blood examinations already given in Section IV are not such as would lend support to the probability of any bacterium being the cause, while the uniformly negative results of blood culture and the uniform absence of organisms from the sections prove at least that the condition is not a bacteriaemia. As to whether it may be ascribed to a bacterial toxin will next be discussed.

6. *Is the condition due to an Intoxication?*

Obviously, until a definite toxin has been found, which, introduced in a pure state, will produce the symptoms of this disease, any positive assertion that the condition resulted from an intoxication would be premature. Nevertheless, it would appear that an 'Intoxication theory' will best fit the facts, so far as they are known at present.

Taking first the question of the food of those who were attacked. At ordinary times, as has been already stated, the food of the native consists of yam, breadfruit, cocoa, peas, beans, cornmeal, salt-fish, and so on. At the time of this epidemic those who were attacked were working in the cane-fields, and during this period of cutting and carrying cane the labourers live almost exclusively on the canes. Many of the natives also, when not actually working

in the cane-fields, live largely on the sugar cane if they can procure it.

Let us presume for a moment that the canes are at fault, and see what can be urged on both sides. Many of the labourers under similar circumstances, working on the same estates, also ate cane and did not suffer. This point is not an insuperable one; firstly, those who escaped may not have been working there long enough, just as beri-beri may take ninety days to develop on a diet of milled rice. Secondly, canes are eaten largely on other estates and in other countries, but such epidemics have not, to my knowledge, been encountered, or, at all events, reported before, and they are certainly far too striking and serious to be overlooked.

The evidence against the sugar-cane itself—the ordinary healthy cane—amounts to practically nothing, but what about the possibility of something in or on the canes affecting only a certain proportion of them?

This idea started a further train of enquiries, and I am informed that sore mouth is not uncommon in cane workers and cane eaters. Apparently this is not due, at all events in the majority, to wounds caused by the cane fibres, nor to any acidity of the juice.

The top part of the cane contains very little sugar; this is cut off before the canes are carted to the factory, or before the person eats any of the cane, or he may break it off with his hands. This top part which is cut or torn off is surrounded by a fine hair-like growth, almost like a powder or fine dust. In some Barbadoes canes these hairs are larger and more irritating, and on this estate near Spanish Town there is, I am told, a larger proportion of these canes than on other estates. This fact is mentioned incidentally; it may have nothing to do with the case. These hairs stick to the fingers and are distinctly irritating, and it is these which appear to cause the sore mouth. The irritation produced by handling the canes is a well-recognised thing, so much so that many of the labourers wear protective covering on the hands and forearms while at work in the fields.

It would be quite a plausible explanation to suggest that in the cutting, and still more in breaking off these tops, the hands become covered with these particles, especially when the labourers are perspiring in the heat of the day. From the hands they would

then be conveyed to the eyes, as when wiping the perspiration from their faces, or to their mouths when eating. The more delicate conjunctival mucous membrane would react more rapidly than the oral, and thus might be explained the 'itching and burning' of the eyes being the first symptom and the sore mouth the next.

Two possibilities now arise, in fact three, but the one can be put out of court very briefly. These possibilities are:—

1. Are these particles themselves the source of a toxin?
2. Are the sores produced the first lesion, as actually part of the disease?
3. Do the sores merely act as a favourable site at which an organism may enter and multiply, or at which a poison may be introduced?

1. *Are the particles themselves the source of the toxin?*

This question, in my opinion, can be ruled out for the following reason. These particles are on every sucker, and if they were the *causa causans* hardly a single labourer on the estates could escape. Cases of sore eyes, sore mouth, and 'peripheral neuritis' are fairly common, but if the above were true they would be almost universal in cane districts.

2. *Whether the sore eyes constitute the primary lesion proper of the disease cannot be stated decisively at present.*

In favour of the positive is the fact that the sequence was, with one exception, the same; 'itching and sore eyes' followed by 'soreness of the mouth'; whereas almost, if not quite as potent an argument against it, is the fact that the eyelids were just as likely to be the first to come into contact with infected fingers when the perspiration was being wiped away from the eyes when the labourers were working under a tropical sun.

3. *As regards the last:*

Such open sores would form an ideal site for the development of organisms. The reasons against the condition being bacterial have already been given, but those arguments apply mainly against the bacteriaemic idea; it is possible that organisms might settle there, and produce their poison which would be absorbed from such an extensive raw surface, just as the diphtheria toxin from a local growth of the Klebs-Löffler bacillus, and like the latter be able to

produce severe nerve lesions, which may leave residual symptoms. The poison in the Spanish Town cases, judging from the post-mortem findings, so affected the nerve tissues as to show clearly the reason why complete recovery is so rare.

Lastly, it is quite plausible to regard the eye and mouth symptoms as being set up by the irritation, as already mentioned, and the other conditions, intestinal and nervous, as due to some ingested toxin, comparable, for example, to ergotism. Such a theory would account for the initial symptoms of sore eyes and mouth and for the two sub-divisions of cases later, those with intestinal symptoms corresponding to the irritation of acute ergotism, and due to local action, and those with later development of nervous symptoms when the poison is absorbed. I do not mean to imply that the poison itself has an action like that of ergot, but merely the analogy of a foreign growth producing intestinal and nervous symptoms.

All that we are justified in saying in our present state of knowledge is that the history, course, and post-mortem findings in the Spanish Town epidemic and in the (wrongly) so-called peripheral neuritis cases indicate that the condition is that of a 'Central Neuritis' due to some toxin, possibly microbial, more probably not, affecting mainly workers on sugar estates, and, again, possibly due to the growth of some fungus or parasite upon the suckers, tops or leaves, of the canes.

VIII. SUMMARY

1. A certain epidemic broke out in the earlier months of 1917 among the labourers on a sugar estate in Jamaica.

2. The onset in each case was sudden, the patients being attacked while at work and apparently in good health.

3. The initial symptoms in all cases were conjunctivitis and stomatitis.

4. Thereafter the patients could be readily divided into two categories: (i) with intestinal symptoms; (ii) with nervous symptoms.

5. The diet of those affected consisted exclusively, or almost exclusively, of sugar-cane.

6. The cane-tops, which are cut or broken off, are covered with

small hairs which are very irritating and may have set up the original conjunctivitis and stomatitis, and, when swallowed, the subsequent diarrhoea.

7. Fresh cases ceased with the cessation of the crop or almost immediately after.

8. No case with early diarrhoea exhibited any affection of the nervous system.

9. Nervous system cases were always constipated until the final two or three days before death.

10. Wassermann reactions with both the blood-serum and the cerebro-spinal fluid were invariably negative.

11. Blood examinations reveal very little abnormality as regards total counts; differential leucocyte counts showed in all cases a marked relative lymphocytosis.

12. Arneth index was very little different from what is found normally in natives in the tropics.

13. The morbid anatomy of the nervous cases is typical of a 'Central Neuritis.'

14. There is no reason for thinking that the disease is pellagral in nature, or has any relation with pellagra.

15. There is no reason for regarding it as beri-beri.

16. There are many contraindications to the condition being a new form of 'deficiency disease.'

17. There is every reason for considering these cases as representing the acute form, or acute stage, of what has for many years been erroneously spoken of as 'Peripheral Neuritis' in Jamaica.

18. There is no positive evidence that the disease is microbial in origin, at least not a bacteriaemia.

19. All the signs and symptoms tend to point to its being a condition of '*Intoxication*.'

December, 1917.

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TAB

Summary of Blo

A = Ankylostome. a. = Ascaris. T = Trichiuris. M = Mala

No.	Initials	Character of Case	Stage of disease	Polymorphonuclear neutrophile	Promyelocyte	Myelocyte	Metamyelocyte	Eosinophile
				%	%	%	%	%
1	D. J.	Mild, intestinal	Height	54·4	...	1·0	0·2	..
2	,,	,, ...	,, ...	43·0	0·2	1·2	...	0·
3	,,	,, ...	Nearly recovered	36·6	0·4	0·8	0·6	0·
4	J. S.	,, ...	Early	46·4	1·2	2·2	1·0	..
5	,,	,, ...	Height	47·2	0·6	2·8	1·6	..
6	J. A.	Severe, intestinal	,, ...	56·6	1·0	1·2	1·2	..
7	E. S.	Average nervous, recovery ...	,, ...	27·2	0·8	0·8	0·6	..
8	V. McC.	,,	Nearly recovered	37·4	0·8	1·6	1·2	..
9	A. B.	Moderately severe nervous ...	Recovering	33·8	0·8	1·2	1·0	..
10	E. M.	Severe nervous	Height	37·4	0·4	1·6	0·6	..
11	,,	,, ...	Same condition	41·2	0·4	0·8	0·6	..
12	C. S.	,, ...	Height	43·0	0·2	0·8	0·8	..
13	R. H.	,, ...	Improving	30·6	0·4	0·8	0·2	.
14	E. G.	Severe and chronic nervous ...	Near height	37·8	0·6	2·0	0·8	0
15	,,	,, ...	Height	46·4	0·8	2·4	1·6	0
16	,,	,, ...	Chronic	49·0	0·8	1·4	1·2	.
17	C. H.	,, ...	Slight improvement after height	48 2	0·6	1·4	1·4	.
18	A. D.	Severe nervous, probably fatal ...	Height	52·8	1·4	1 0	1·0	.
19	J. P.	Severe, fatal, nervous	Early	45·0	0·4	0·6	1·0	.
20	,,	,, ...	Height	41·0	0·6	1·4	0·2	0
21	,,	,, ...	In extremis	64·0	1·0	1·2	...	.
22	G. P.	,, ...	Near height	47 8	0·2	0·2	0·8	
*23	,,	,, ...	Near end	67·0	0·2	0·8	...	
24	T. J.	,, ...	At height	49·8	0·2	0·4	0·8	
25	,,	,, ...	In extremis	54·6	0·8	0·6	1·0	
26	M. H.	,, ...	Near height	60·4	0·2	0·6	0·2	
27	J. N.	Very chronic; *see* case	Stationary	41·6	0·6	1·8	0·4	
28	S. C. T.	,, ,, ...	,, ...	37·6	0·8	0·4	0·2	

aminations.

= Amoeba of Dysentery. Pyocy. = *B. pyocyaneus.*

Eosinophile	Basophile	Large Mononuclear	Transitional	Large Lymphocyte	Small Lymphocyte	Türk's cells	Rieder cells	Arneth Count						Arneth Index		
								I	II	III	IV	V	Stabkernige	I and II	I, II and half III	
%	%	%	%	%	%	%	%									
·8	0·4	1·0	0·6	4·6	32·8	...	1·2	9·9	40·4	32·4	12·2	2·2	2·9	50·3	66·5	A. a. T.
·2	0·8	3·6	1·6	7·6	29·8	1·0	1·6	3 7	36·4	35·3	13·9	3·2	7·5	40·1	57·7	
·8	2·4	4·8	3·8	7·4	30·4	0·2	2·4	6·5	37·1	32·2	13·8	3·6	6·8	43·6	59·7	
·2	1·4	4·0	2·4	5·4	30·8	0·8	2·2	15·1	41·4	25·8	8·2	2·2	4·3	59·5	72·4	M. D. Pyocy.
·4	1·2	3·6	0.8	6·0	31·4	1·0	3·4	13·6	50·4	22·0	7·6	1·3	5·1	64·0	75·0	
·2	1·8	3·0	2·2	6·2	21·2	0·6	1·8	17·0	40·9	25·5	7·4	5·0	4·2	57·9	70·6	
·0	0·6	3·2	1·6	7·4	49·8	...	3·0	6·6	38·9	29·5	16·9	4·4	3·7	45·5	60·2	A. T.
2	0·4	4·6	2·8	7·2	33·8	1·8	0·2	6·4	42·8	38·0	4·8	1·1	6·9	49 2	68·2	A.
·0	0·6	6·6	4·6	9·2	21·8	1·8	0·6	3·0	39·1	37·3	11·8	3·5	5·3	42·1	60·7	M. A.
·2	0·6	4·6	1·8	9·8	35 6	1·0	2·4	9·1	53·0	21·4	9·6	3·2	3·7	62·1	72·8	
·0	1·2	3·8	2·8	8·4	33·2	1·6	2·0	5·8	47·1	35·4	7·8	1·5	2·4	52 9	70·6	
0	1·8	3·6	2·4	11·6	29·6	1·4	0·8	6·5	42·3	25·6	13·0	3·7	8·9	48·8	61·6	
4	0·4	2·2	1·0	8·6	39·0	1·0	0·4	6·6	45·7	27·5	12·4	1·9	5·9	52·3	66·05	A. a.
0	1·8	3·4	3·4	9·8	28·4	0·4	1·2	5·8	48·7	31·2	5·3	2·6	6·4	54·5	71·1	A.
4	0·8	4·6	3·4	10·2	18·0	2·6	0·6	8·2	46·1	28·9	7·3	3·5	6·0	54·3	68·7	
8	0 2	4·2	2·2	7·0	26·2	1·2	0·8	11·4	58·0	19·2	2·0	1·6	7·8	69·4	79·0	
2	1·6	2·4	3·2	9·6	24·6	3·4	0·4	8·7	46·5	30·3	7·0	4·6	2·9	55·2	70·8	
0	0·6	2·6	1·6	7·6	28·2	1·0	0·2	10·3	42·0	29·9	11·0	3·4	3·4	52·3	67·2	A. a.
0	0·6	2·6	2·2	7·4	35·2	1·0	...	4·5	33·8	40·0	12·5	5·4	3·8	38·3	58·3	A.
2	0·8	2·2	1·8	5·4	35·8	2·8	0·4	6·8	36·6	36·1	12·7	3·4	4·4	45·4	61·4	
8	1·0	3·2	2·6	3·2	14·6	2·2	0·2	6·6	43·1	30·6	9·7	6·9	3·1	49·7	65·0	
4	0·4	3·2	3·6	9·6	28·8	2·6	1·4	5·4	38·5	35·6	12·9	5·5	2·1	43·9	61·7	
0	1·0	3·4	1·2	4·6	17·8	2·0	...	7·7	43·0	33·4	9·6	4·8	1·5	50·7	67·4	
0	1·2	2·0	2·0	6·2	32·2	1·8	0·4	8·4	53·4	24·6	8·4	2·4	2·8	61·8	74·1	A. a.
8	1·4	2·4	2·2	5·0	25·0	1·2	1·0	8·7	54·6	23·9	8·1	1·8	2·9	63·3	75·2	
2	1·2	5·8	3·0	6·6	17·8	0·4	0·6	8·9	46·4	27·5	11·3	3·3	2·6	55·3	69·0	
6	2·0	4·4	2·2	10·8	32·8	1·0	0·8	6·3	35·1	32·7	14·0	6·3	5·6	41·4	57·7	
0	2·2	3·0	4·2	12·0	26·6	1·8	1·0	2·7	26·1	34·0	25·0	5·3	6·9	28·8	45·8	A.

TA

Summary of Sympt

N = Normal. + = present. — = absent or unaffected. ? = doubtful. x = variable. ... = not tes

		D. J.	J. S.	J. A.	C. D.	H. B.	E. S.	V. McC.	A. B.	E. N
	Intellect	N	N	N	N	N	N	N	N	N
Onset	Sore eyes	+	+	+	–	+	+	?	+	+
Onset	Sore mouth	+	+	+	+	+	–	+	+	+
	Diarrhoea early	+	+	+	–	–	–	–	–	–
	,, terminal	–	–	...	+	–	–	–	–	–
	Constipation	–	–	–	+	+	+	+	+	+
Numbness	Feet	–	–	–	+	+	+	+	+	+
Numbness	Legs	–	–	–	+	+	+	+	+	+
Numbness	Thighs	–	–	–	+	–	?	–	+	±
Numbness	Hands	–	–	–	–	–	–	–	+	+
Numbness	Arms	–	–	–	–	–	–	–	–	±
Numbness	Tongue	–	–	–	–	–	–	–	–	–
	Dysarthria	–	–	–	–	–	–	–	–	–
Changes in or abnormalities of sensation	Girdle	–	–	–	...	–	+	–	–	+
Changes in or abnormalities of sensation	Touch	–	–	–	...	–	–	–	–	–
Changes in or abnormalities of sensation	Heat and cold	–	–	–	...	+	...	...	...	–
Changes in or abnormalities of sensation	Pain	–	–	–	...	+ ?	–	–	–	–
Changes in or abnormalities of sensation	Position	–	–	–	...	–	–	–	–	–
Changes in or abnormalities of sensation	Joint	–	–	–	...	–	–	–	–	–
Changes in or abnormalities of sensation	Muscle pressure	–	–	–	...	–	–	–	–	–
Changes in or abnormalities of sensation	Formication	–	–	–	+	–	–	–	–	–
Changes in or abnormalities of sensation	Weights	–	–	–	...	–	–	–	–	–
Changes in or abnormalities of sensation	Vibration	–	–	–	...	–	...	–	–	–
Changes in or abnormalities of sensation	Localisation of sensation ...	N	N	N	...	N	N	N	N	N
	Stereognosis	N	N	N	...	N	N	N	N	N
	Paralysis	–	–	–	...	–	–	–	–	–
	Atrophy of muscle	–	–	–	...	–	–	–	–	

ented.

weakness (in place of 'numbness.') ± = affected, but in slight degree only.

. S.	R. H.	E. G.	C. H.	A. D.	J. P.	G. P.	T. J.	M. H.	J. M.	J. N.	S. C. T.
N	N	N	N	N	N	N	N	Fair	N	N	N
+	+	+	+	+	+	+	+	+	+	+	−
+	+	+	−	+	+	+	+	+	+	+	−
−	−	−	−	−	−	−	−	−	−	−	−
−	−	−	−	−	+	+	+	+	+	−	−
+	+	+	+	+	+	+	+	+	+	+	x
+	+	+	+	+	+	+	+	+	+	+	+cv
+	+	+	+	+	+	+	+	+	+	+	+cv
−	+	+	+	+	±	+	−	±	+	+	−
−	+	+	+	+	+	+	+	+	−	+	+cv
−	−	−	−	+	+	+	+	+	−	−	—
−	−	−	−	−	+	+	+	+	−	−	−
−	−	−	−	−	+	+	+	?	−	−	−
+	+	−	−	−	−	−	−	+	−	−	−
−	−	±	−	−	−	−	−	−	−	−	−
−	+	delay	...	delay	−	+	...	−	−	−	+
−	−	−	+ ?	−	−	−	+ ?	−	−	−	−
−	−	+ ?	−	+	−	+	+	−	+	−	−
−	−	+ ?	−	+	+	±	+	−	+	−	−
−	−	−	−	− ?	+	−	−	−	−	−	−
−	−	+	+	−	−	−	−	−	−	−	−
...	...	+	...	...	−later+	...	...	...	−	−	−
−	−	−	−	−	−	−	−	−	−	−	−
N	N	N	N	N	N	N	N	N	N	N	N
N	...	−	N	...	...	−	?	...	N	N	N
−	−	−	−	−	−	−	—	−	−	−	−
−	−	−	−	−	general emacia-tion	general emacia-tion	−	−	−	−	−

TABL

Summary of Sympton

N = Normal. + = present. − = absent or unaffected. ? = doubtful. x = variable. ... = not teste

		D. J.	J. S.	J. A.	C. D.	H. B.	E. S.	V. McC.	A. B.	E. M.
Gait	Steppage	−	−	−	...	−	−	−	+	−
	Spastic...	−	−	−	...	−	−	−	−	±
	Ataxic	−	−	−	...	−	+	−	−	−
	Tabetic	−	−	−	...	−	−	−	−	−
	Not characteristic but abnormal	−	−	−	...	+	−	−	−	+
Incoordination	Romberg	−	−	−	...	−	?	−	−	−
	Legs: out of bed	−	−	−	...	−	+	−	−	+
	in bed	−	−	−	...	−	−	−	−	±
	Hands	−	−	−	...	−	−	−	−	+
Pupils	Light reaction	N	N	N	...	N	N	N	N	N
	Convergence	N	N	N	...	N	N	slow	N	N
	'Dark Vision'	−	−	−	...	−	−	+	+	−
	Nystagmus	−	−	−	...	−	−	−	−	−
	Ocular paresis	−	−	−	...	−	−	−	−	−
	Diplopia	−	−	−	...	−	−	−	−	−
	Vertigo	−	−	−	...	−	−	−	−	−
	Deafness	−	−	−	...	−	−	−	−	−
	Taste	N	N	N	...	N	N	N	N	N
Reflexes	Jaw	N	N	N	...	N	N	N	N	N
	Elbow	N	N	N	...	N	N	N	N	N
	Wrist	N	N	N	...	N	N	N	N	?
	Knee	N	N	N	...	N	−	?	−	−
	Ankle	N	N	N	...	N	−	?	−	−
	Babinski	N	N	N	...	N	−	?	?	−
	Superficial	N	N	N	...	N	x	?	N	x
	Sphincters	N	N	N	...	N	N	?	N	N
	C. S. Fluid	...	...	N	...	...	...	...	...	N

–continued.

sented.

= weakness (in place of 'numbness'). ± = affected, but in slight degree only.

C. S.	R. H.	E. G.	C. H.	A. D.	J. P.	G. P.	T. J.	M. H.	J. M.	J. N.	S. C. T.
+	–	...	...	...	–	–	–	–	–	+	–
–	+	...	...	...	–	–	–	+	+	+	±
–	–	...	...	...	–	+	+	+	+	–	–
–	–	...	...	...	–	–	–	–	–	–	–
–	–	...	...	...	+	–	–	–	–	–	–
–	–	...	...	...	–	?	–	–	...	–	–
±	...	+	...	...	+	+	+	+	+	–	–
–	–	±	+	+	±	–	–	±	±	...	...
–	–	+	+	+	+	+	+	+	–	–	–
N	N	N	N	N	N	N	N	N	N	N	N
N	N	N	N	N	N	N	N	N	N	N	N
–	+	+	–	–	–	–	–	–	–	–	–
–	–	–	–	–	–	–	–	–	–	–	–
–	–	–	–	–	–	–	–	–	–	–	–
–	–	–	–	–	–	–	–	–	–	–	–
–	–	–	–	–	–	–	–	–	–	–	–
–	±	–	–	–	–	±	–	–	–	–	–
	N	N	N	N	N	N	N	N	N	N	N
	N	N	N	N	N	N	...	N	N	N	N
	N	–	N	N	?	?	N	N	N	N	N
	?	–	?	–	–	–	–	N	N	N	N
	–	–	–	–	–	–	–	+later–	–	+	+ ?
	–	–	–	–	–	–	–	+later–	–	+	...
	...	N	...	?	?	...	–	?	...	–	?
	N	N	N	N ?	N	x	...	N	N	N	N
	N	N	N	N	N	N	N	N	N	N	N
	...	...	...	...	N	N	N	...	...	...	...

EXPLANATION OF PLATES

Plate II

Fig. 1. Lumbar enlargement, lower part.

Fig. 2. Lumbar enlargement, upper part.

Plate III

Fig. 3. Lower dorsal cord.

Fig. 4. Upper dorsal cord.

Plate IV

Fig. 5. Hypophysis, $\frac{2}{3}$-in.

Fig. 6. Cells of Clarke's column, $\frac{2}{3}$-in.

Plate V

Fig. 7. Optic nerve, $\frac{2}{3}$-in.

Fig. 8. Optic nerve, $\frac{1}{6}$-in.

Plate VI

Fig. 9. Nerve fibres at posterior root ganglion, $\frac{1}{6}$-in.

Fig. 10. Tibialis anticus, $\frac{1}{6}$-in.

Plate VII

Fig. 11. Pancreas, $\frac{1}{6}$-in.

Fig. 12. Liver, $\frac{1}{6}$-in.

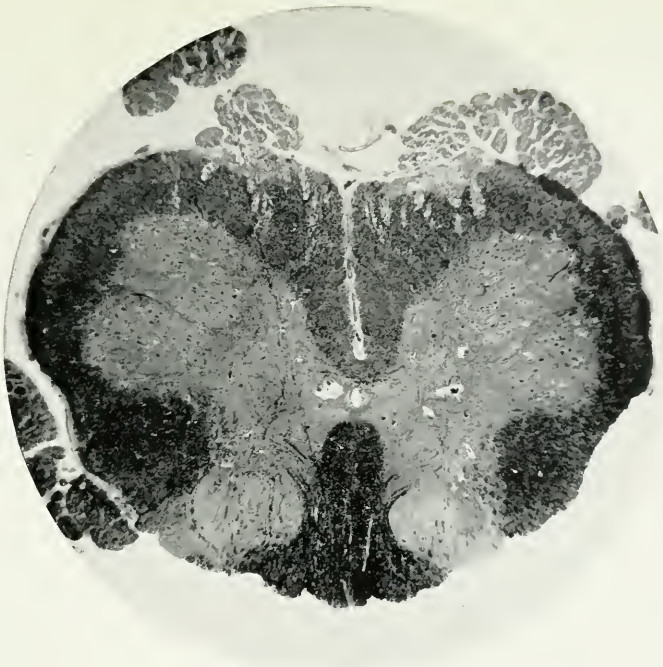

Fig. 1

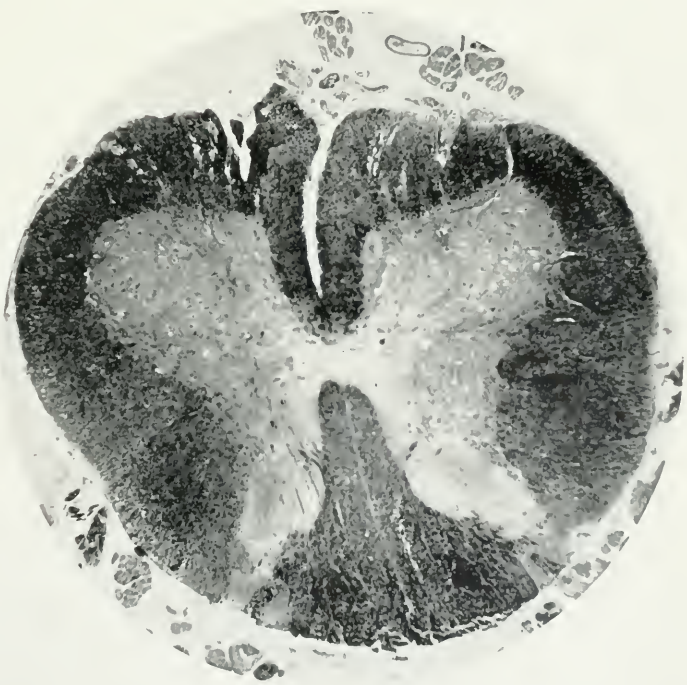

Fig. 2

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Fig. 3

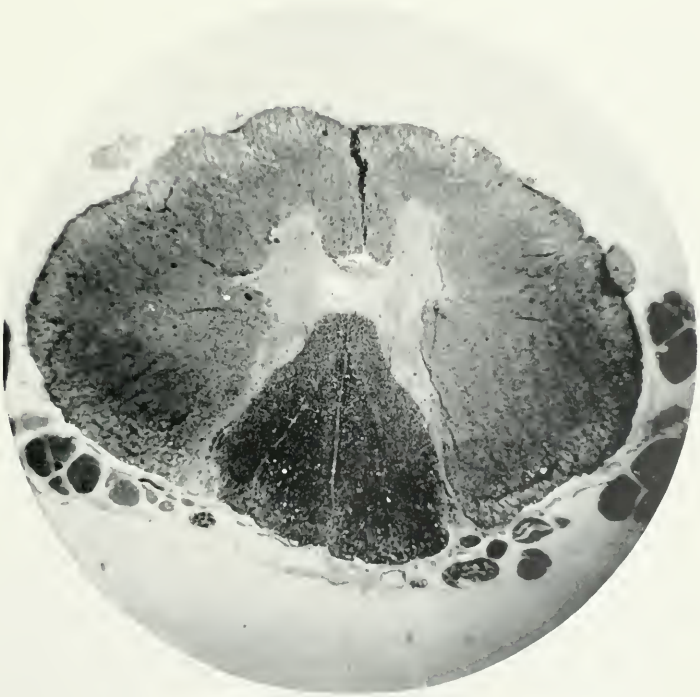

Fig. 4

C. Tinling & Co., Ltd., Imp.

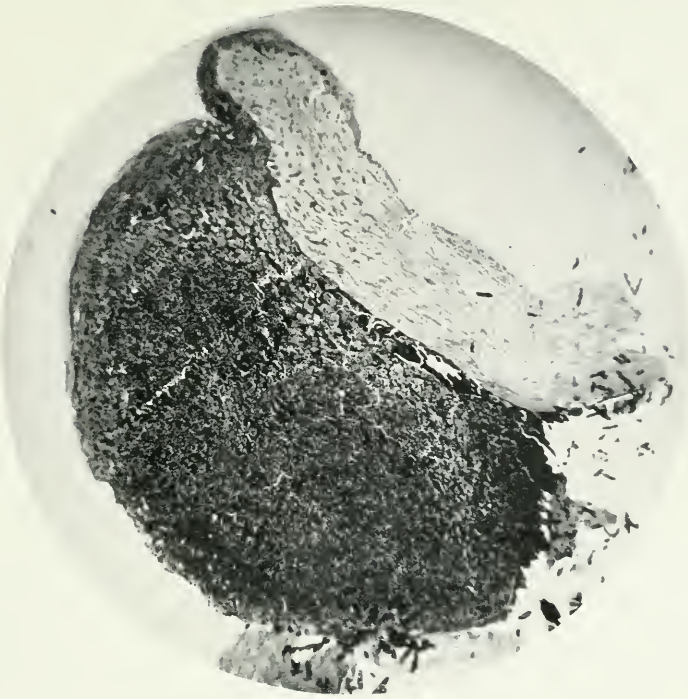

Fig. 5

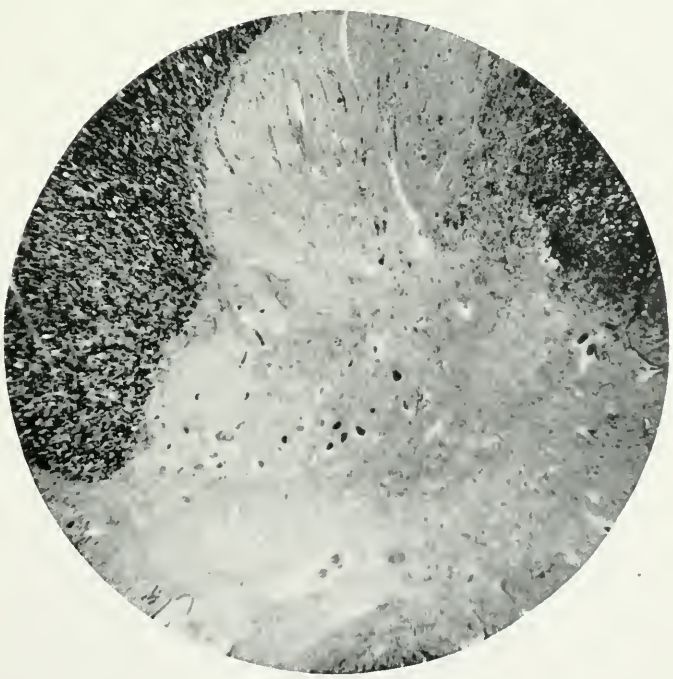

Fig. 6

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Fig. 7

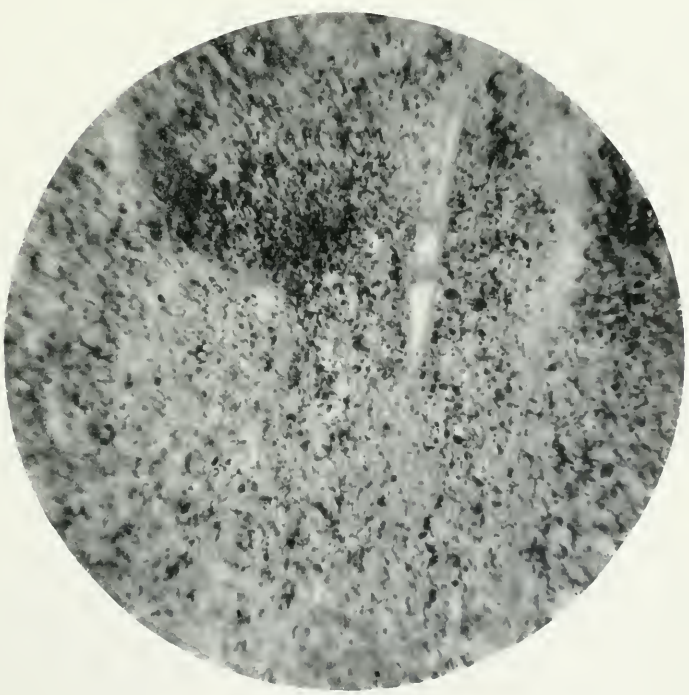

Fig. 8

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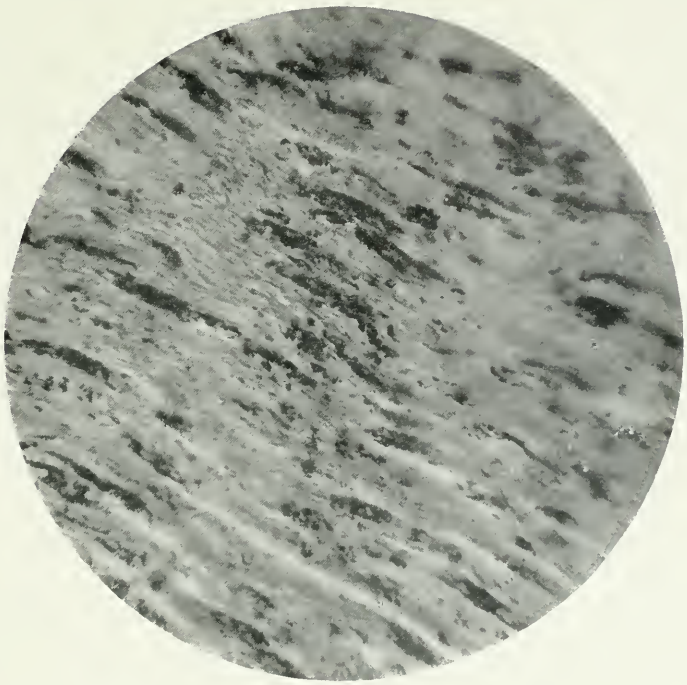

Fig. 9

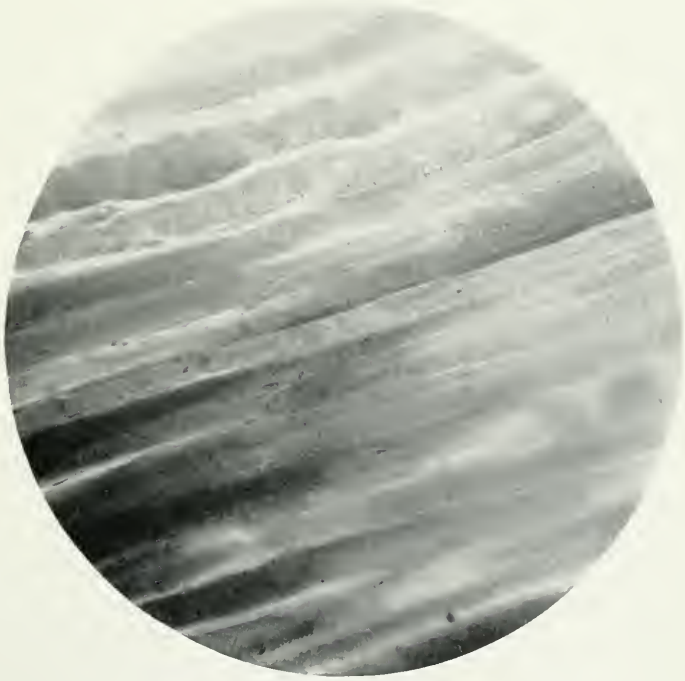

Fig. 10

C. Tinling & Co., Ltd., Imp.

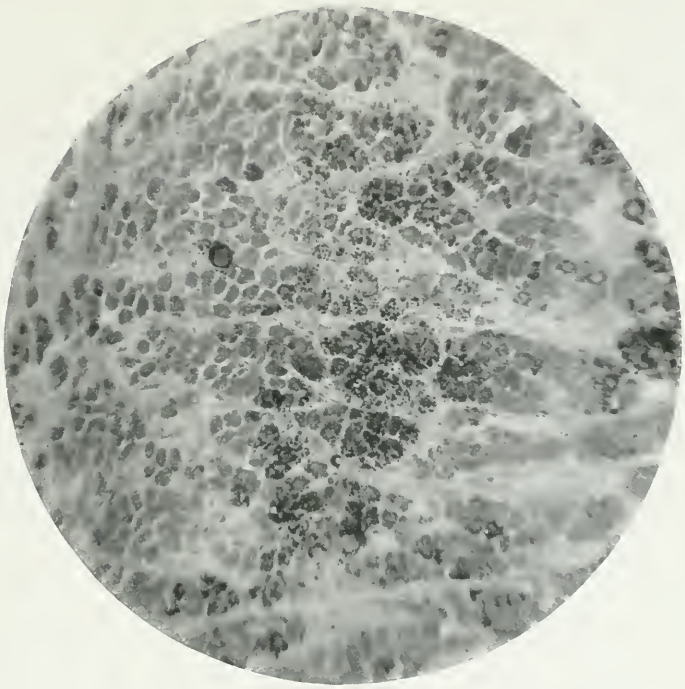

Fig. 11

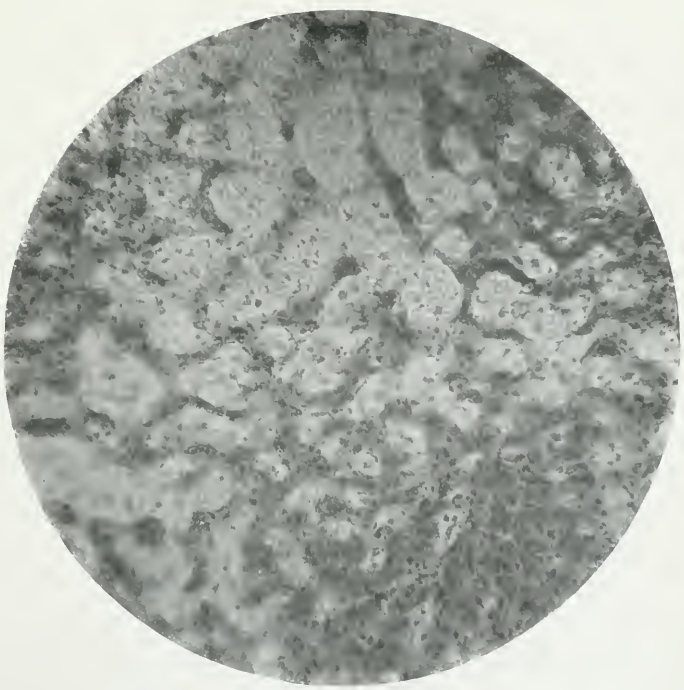

Fig. 12

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